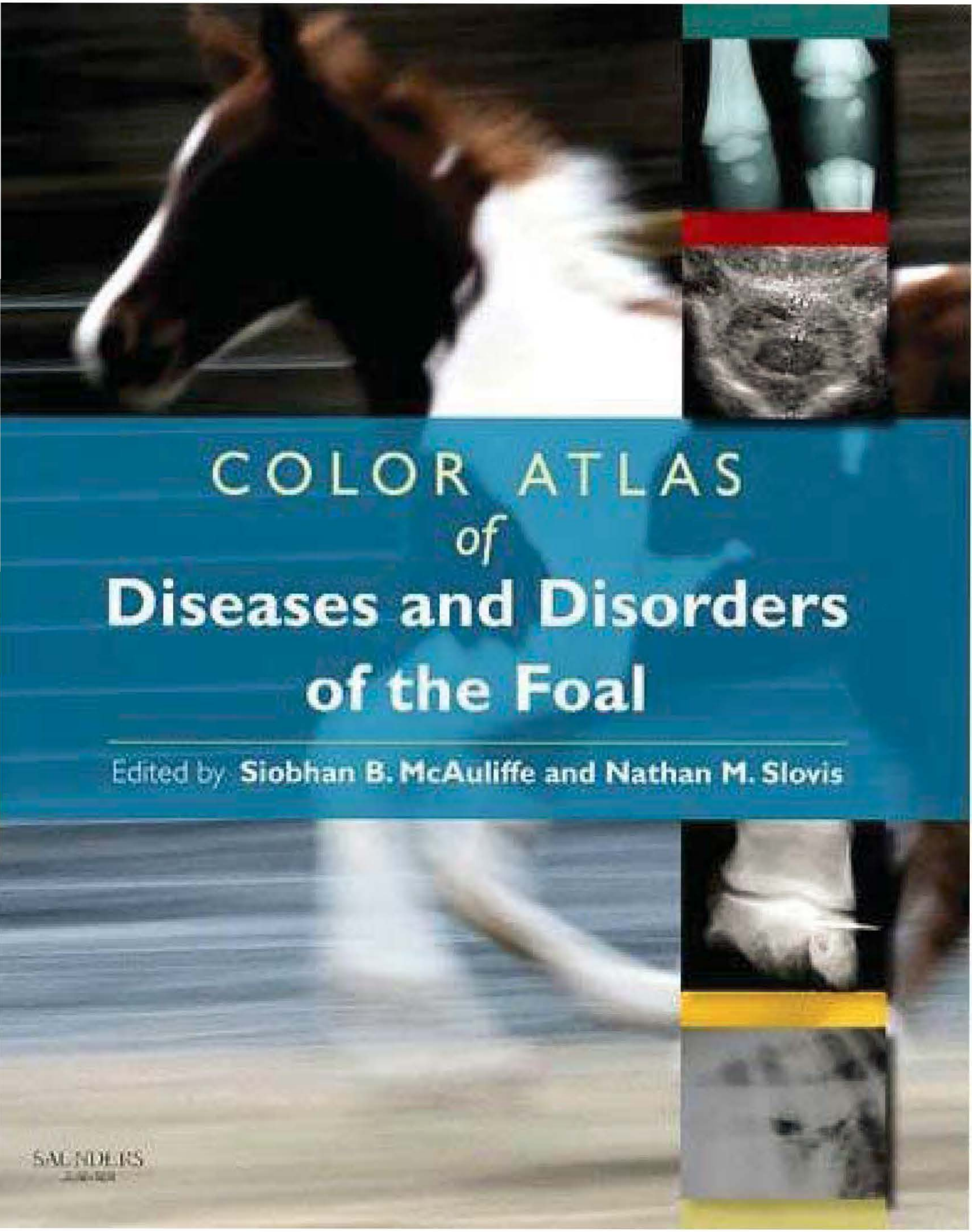
COLOR ATLAS
of
Diseases and Disorders
of the Foal
Edited by: Siobhan B. McAuliffe and Nathan M. Slovis
SAUNDERS

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ELSEVIER

An imprint of Elsevier Limited

First published 2008

ISBN 978 0 7020 2810 6

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

Library of Congress Cataloging in Publication Data
A catalog record for this book is available from the Library of Congress

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The Publisher

Printed in China

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PREFACE

Today's veterinary curriculum places more demands on both students and teachers and consequently there is generally insufficient time available to teach all aspects of an expanding knowledge of equine pediatric medicine and surgery.

The purpose of this atlas is to provide both the general practitioner and student with a reference guide. The atlas's emphasis is on normal and abnormal findings of different body systems examined by the general equine practitioner. It contains excellent digital images and illustrations that provide a base of knowledge on which the reader may build and describes techniques involved in neonatal and pediatric medicine and surgery, so as to make learning easier and more enjoyable.

This atlas provides clear, easily accessible information on history, clinical signs, differentials, treatment and prognosis. It should be regarded as a companion to other equine texts that have educated the reader on the pathophysiology of the various diseases illustrated in the atlas.

We have attempted to emphasize the diagnostic features of those conditions the general equine practitioner will be frequently exposed to but have included some less common conditions for completeness and interest.

Through this book we have oriented many years of clinical experience in pediatric medicine and surgery by leading specialists. The editors realized that the most valuable learning resources they possessed were the photographs that they took of their patients. These photographs and schematics will provide the reader with a large range of images from which to learn and compare, while helping the practitioner gain confidence in equine pediatric medicine and surgery.

The chapters are organized according to the body system being discussed. Each chapter starts by discussing diagnostic techniques associated with the given body system accompanied by photographic and, where appropriate, schematic representations of normal features. The abnormal findings are then discussed as either congential or acquired disorders. Acquired disorders are then further broken down and discussed under various subheadings. We have strived to include as many images as possible of each disorder so that the reader can be guided from presentation, through diagnosis and differentials to the outcome of the case. We have included case histories and outcomes in many of the image legends and we hope this is beneficial to the reader.

Significant progress has been made in equine pediatric medicine over the last two decades and today the public demands more specialized care for their animals. We hope that the images in the following pages stimulate interest in further learning and aid in the timely diagnosis of many foal disorders.

Siobhan McAuliffe Nathan Slovis

ACKNOWLEDGEMENTS

Putting together a well-illustrated book obviously requires both text and illustrations. Our thanks must first go to all of those who contributed chapters to this book. They have given of their expertise and time with little in return other than harassing e-mails and the knowledge that they have yet again helped to educate others. We have tried to use as many practitioners as possible when compiling the text in order to give an emphasis to those disorders that are more commonly encountered and to present the information in a way in which it is easily accessible. This has however meant that we asked some very busy people to contribute for us. We cannot adequately state how appreciative we are of the time they have dedicated to our endeavour and the enthusiasm they have shown throughout.

A special thanks is extended to Dr. Ursula Fogarty, Dr. Stefania Bucca and Dr. Brendan Farelly, who kindly asked us to include their new information on atlanto-occipital ultrasound.

Obtaining the photographic images for this book has taken many years. We cannot state how grateful we are to those who generously provided us with these images. In particular Dr. Monica Aleman and Dr. Genevieve Fontaine-Rodgerson provided us with a large selection from which to choose. A further thanks has to be extended to Dr. Ursula Fogarty and Dr. Celia Marr, who patiently answered questions and provided legends for their images.

Our thanks and appreciation also goes to the staff and management of the McGee Medical center and The Stables of King Abdullah and Sons. Their patience and tolerance with our amateur photography has resulted in many of the images on the following pages.

The illustration team at Elsevier must be congratulated on the magnificent job they have done with the drawings throughout. The support staff at Elsevier have shown endless patience and understanding from start to finish.

Finally we would like to thank out spouses, Fernando and Stacey for their support and understanding in the four years that it has taken us to put this book together; and our children, Fernando and Riley for providing us with constant inspiration and joy.

CHAPTER 1

The pregnant mare

Walter Zent DVM and Lucas Pantaleon MV, MS, DACVIM

Introduction

The breeding success of a mare can no longer be judged on her ability to conceive alone but rather on her ability to produce a live, viable foal. Advances in theriogenology have resulted in high conception rates and improvements in neonatal medicine have resulted in greater survival rates of foals. However disease processes are often advanced when presented as they may have started in utero. Awareness of an existing problem prior to parturition facilitates early intervention and treatment and consequently will aid in decreasing perinatal deaths which account for a significant percentage of foal mortality. Increasing awareness of fetal wellbeing and the impact that it has on neonatal viability has led to closer monitoring of the peripartum mare and improved techniques in fetal monitoring.

Complications in late gestation

It is estimated that 10–15% of mares undergo embryonic or fetal loss at some point in gestation. Most occurs in the first 40 days of pregnancy, when the primary corpus luteum is the sole source of progesterone. Thus, once confirmed to be 45–60 days pregnant most mares can be expected to carry the fetus to term. However, abortions that occur during the last trimester can be particularly stressful for the owner as there is a sense of being so near and yet so far. Every effort should be made to diagnose the cause of an abortion as some agents such as equine herpes virus may occur in storms and preventative measures may be taken to limit further loss.

In addition to fetal loss through abortions, life-threatening situations for the mare may develop in late gestation such as ventral body wall ruptures, uterine torsion and hydrops of the fetal membranes. Accurate diagnosis and timely intervention may not only save the life of the mare but may also result in the birth of a viable foal in cases of ventral body wall ruptures and uterine torsions. This chapter reviews some of these late gestation complications and causes of abortion.

Placentitis (Figs 1.1–1.20)

History

- Placentitis is an important cause of abortion, stillbirth and perinatal death in horses. Different etiologic agents such as viruses (equine viral arteritis and equine herpes virus 1), bacteria (β-hemolytic *Streptococci*, *Escherichia coli*, *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, *Leptospira* spp and *Crossiella equi*) and fungi (*Aspergillus* spp and *Mucor* spp) have been implicated as causative agents.
- Depending on the microorganism involved, several routes of infection are possible:
  - Ascending infections are common, resulting in inflammation and detachment of the chorioallantois at the level of the cervical star.
  - Hematogenous infection of the fetal membranes has also been described. This is the primary route of infection for *Leptospira* spp., which cause a diffuse or multifocal placentitis. Viruses also gain access to the placenta and fetus via the hematogenous route.
  - A unique form of placentitis is caused by the bacteria *Crossiella equi* (nocardioform placentitis). It has been suggested that the organism gains access to the mare's uterus during breeding and remains latent until later in gestation. The characteristic lesions are localized at the level of the uterine body.

Clinical signs

- Clinical signs of placentitis may include premature udder enlargement and lactation, cervical relaxation, vaginal discharge, abortion, stillbirth and premature delivery.
- Most mares with placentitis do not become systemically ill and the hemogram remains within normal ranges.
- Placentitis can also result in the birth of immature or dysmature foals, small for gestational age foals or neonatal sepsis.

Figure 1.1: Ascending placentitis viewed from the allantoic surface, note the thickening and discoloration of the placenta around the cervical star.

Figure 1.2: Ascending placentitis, same placenta as Fig 1.1 viewed from the chorionic surface. Note the absence of villi and presence of exudates in the affected area.

Figure 1.3: Ascending placentitis with extensive exudate (courtesy of the Irish Equine Centre – IEC)

Figure 1.4: Fungal placentitis, note the thickened leathery appearance.

A **B**

Figure 1.5: (a) & (b) Fungal placentitis, note the fungal plaques evident on both the allantoic and chorionic surfaces of the placenta from a fresh abortion at 5 months of gestation.

Figure 1.6: *Aspergillus* spp cultured from the placenta in Fig 1.4.

Figure 1.7: Chronic diffuse placentitis with marked thickening of the placenta (courtesy of IEC).

Figure 1.8: Aborted foal at 10 months of gestation (placenta in Fig 1.7). Note the dehydrated appearance of the foal. Foals from mares with chronic placentitis are frequently delivered or aborted with this appearance (courtesy of IEC).

Figure 1.9: *Crossiella equi* placentitis (nocardioform placentitis). Note the necrotic area of placentitis at the junction of the body and horn (courtesy of IEC).

Figure 1.10: *Crossiella equi* placentitis (nocardioform placentitis), same placenta as Fig 1.9 viewed from the allantoic surface (courtesy of IEC).

Figure 1.11: Premature lactation in a mare with ascending placentitis.

Figure 1.12: Discharge of the cervical plug at 9 months of gestation in a mare with ascending placentitis.

Figure 1.14: In utero growth retardation (IUGR) of a foal from a mare that had *Crossiella equi* placentitis (nocardioform placentitis).

Figure 1.13: Vaginal discharge from a mare with cervical placentitis.

Differential diagnosis

Other causes of abortion, stillbirth or premature delivery. Placental changes may not be evident to the naked eye and a complete postmortem examination should be performed on any aborted fetus with samples of placenta and appropriate fetal organ samples taken for histopathology.

Diagnosis

- ***Clinical signs. Any mare with premature udder development or a vaginal discharge should be regarded as having placentitis until proven otherwise.***
- Normal complete cell count and fibrinogen.
- Samples obtained from the vaginal or cervical discharge should be submitted for culture and cytological examination.
- Transrectal ultrasonography is performed in order to image the area at the level of the cervical star. Ultrasonography may show an increase in the combined thickness of the uterus and the placenta (CTUP), edema of the allantochorion and placental separation. The CTUP should be measured in the ventral part of the uterine body as placenta in the dorsal part is often thicker, with edema of the dorsal placenta being found in a number of normal mares in the last month of gestation. The normal CTUP values by gestational age are:

<5 mm	<270 days
<8 mm	271–300 days
<10 mm	310–330 days
<12 mm	330+ days

- Transabdominal ultrasonography is an important tool to assess the placenta at the level of the body where nocardioform placentitis occurs. The mean utero–placental thickness as measured by transabdominal ultrasound should be 11.5 ± 2.4 mm in late gestation. Another very important use of transabdominal ultrasonography is to assess the fetal wellbeing in mares with suspected placentitis. Fetal heart rate (FHR), movement, and growth can be checked and the turbidity of the amniotic fluid can be assessed (see p. 17).

Treatment

- Broad-spectrum antibiotics such as trimethroprim-sulfadiazine or a gentamicin and penicillin combination are good initial choices and have been shown to achieve acceptable levels within the fetal fluids.
- Non-steroidal anti-inflammatory drugs (flunixin meglumine or phenylbutazone) are used with the objective of decreasing the level of inflammatory mediators released during placentitis.

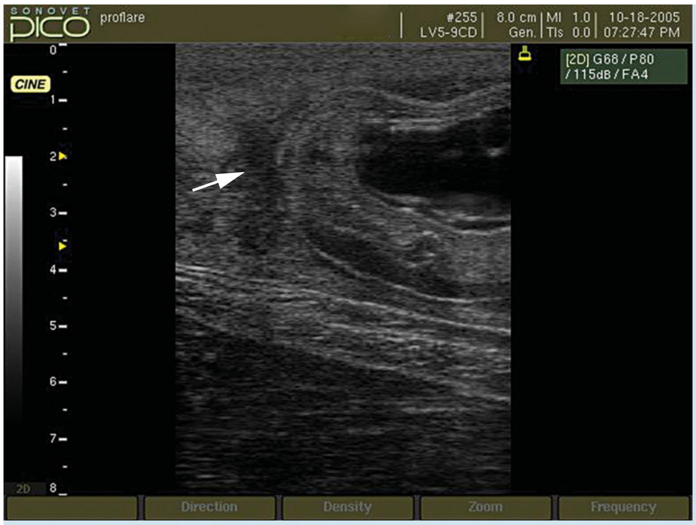

Figure 1.15: Ultrasonographic image of the uteroplacental unit at the area of the cervical star demonstrating thickening and separation (arrow) of the placenta.

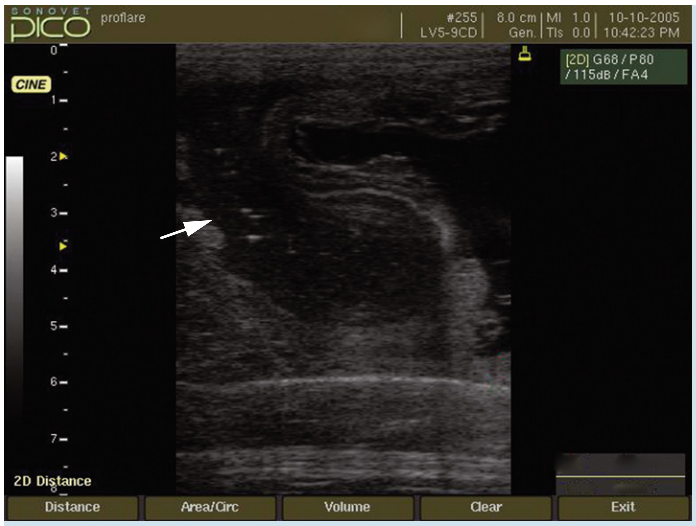

Figure 1.16: Ultrasonographic image of the uteroplacental unit at the area of the cervical star demonstrating thickening and separation (arrow) of the placenta. Note the retroplacental exudates.

Figure 1.17: Same mare as in Fig 1.16 following 3 weeks of therapy. Note that the volume of retroplacental exudate has decreased.

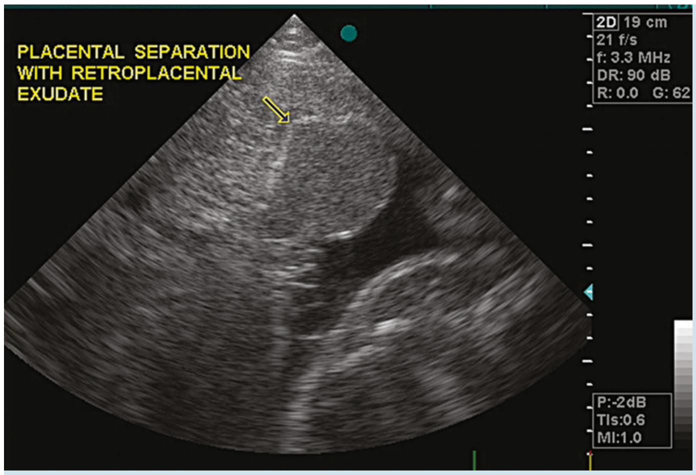

Figure 1.18: Ultrasonographic image of the cervical area demonstrating placental separation with retroplacental exudate. Note the foal in the bottom right hand corner of the image.

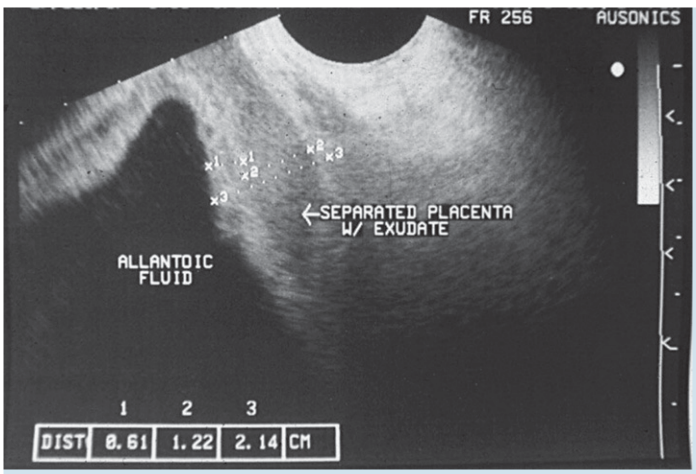

Figure 1.19: Transabdominal ultrasonographic image of the uteroplacental unit demonstrating separation with retroplacental exudates. The measurements are as follows: 1 = placental thickness, 2 = exudate thickness, 3 = thickness of combined uteroplacental unit including exudate.

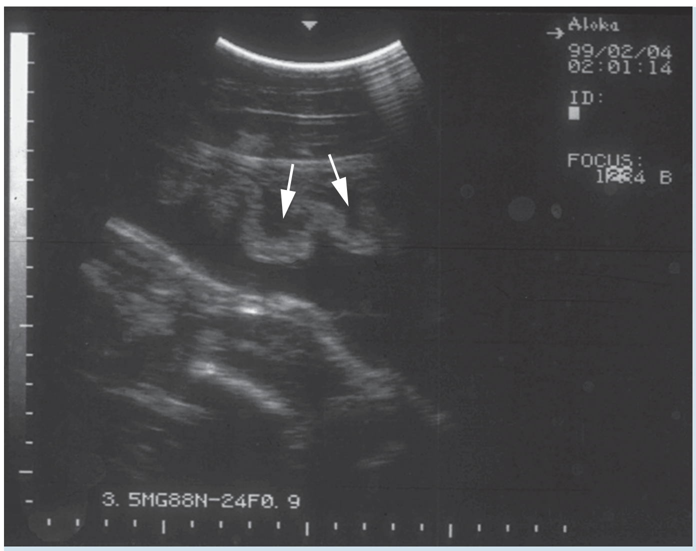

Figure 1.20: Transabdominal ultrasonographic image of the uteroplacental unit demonstrating separation (arrows) and folding of the placenta.

- Altrenogest used at a double dose (0.088 mg/kg every 24 hours) may be of benefit in the treatment of uteroplacental inflammation secondary to placentitis, although this remains controversial. Progestins are also thought to decrease myometrial activity.
- Pentoxifylline and aspirin are other controversial treatments that are thought to improve placental perfusion.

Hydrallantois/Hydramnios (Figs 1.21–1.26)

History

- Hydrallantois is an uncommon condition in the mare that develops during the last trimester of pregnancy and is characterized by the excessive accumulation of allantoic fluid.
- The allantoic fluid volume in mares with hydrallantois can range from 110 to 230 liters, while a normal volume at term ranges from 8 to 18 liters.
- Mild diffuse placentitis or endometrial vasculitis have been implicated as causes of hydrallantois in some mares.
- Hydrops of the amniotic fluid is much less common and is thought to be related to swallowing defects in the fetus.

Clinical signs

- Rapid abdominal enlargement over a period of 1–2 weeks. The mare shows difficulty walking, lying down and standing due to the increased abdominal size.
- Other signs include ventral edema, varying degrees of colic, anorexia, tachycardia and dyspnea, and mucus membranes may appear cyanotic (especially when the mare is lying down).

Differential diagnosis

Other causes of increased abdominal size such as twin pregnancy, body wall hernias and prepubic tendon rupture should be differentiated from hydrops.

- Twin pregnancy causes a gradual abdominal enlargement.
- Ventral abdominal ruptures can be sequelae of hydrallantois, due to the stress on the prepubic tendon and ventral abdominal muscles caused by the weight of fetal fluids.

Diagnosis

- Physical examination: normal rectal temperature, tachycardia, dyspnea, cyanosis, difficulty ambulating, obvious abdominal enlargement and ventral abdominal wall edema.

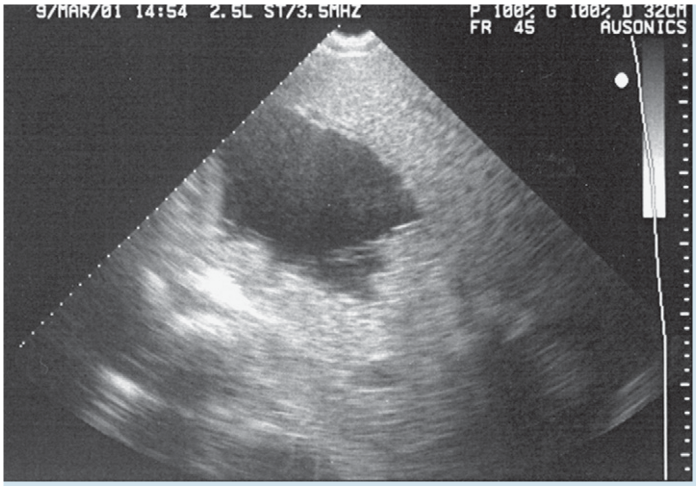

Figure 1.22: Transabdominal ultrasonographic image from a mare with hydrallantois showing the abnormally fluid filled (allantoic) non-pregnant uterine horn.

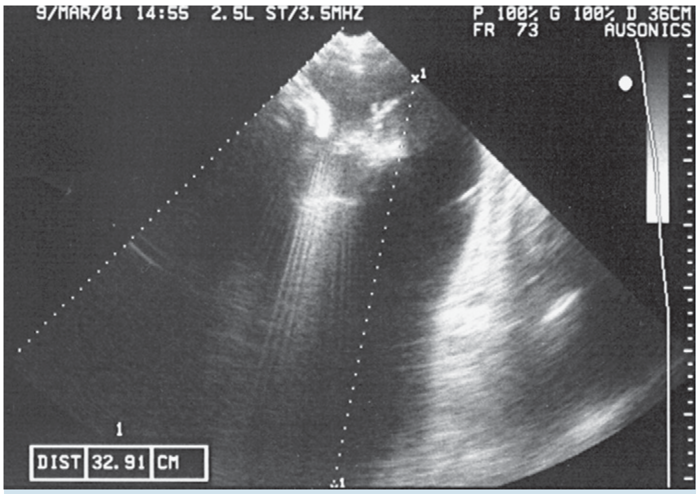

Figure 1.21: Transabdominal ultrasonographic image from a mare with hydrallantois during late gestation. The allantoic fluid depth is 32.91 cm (normal mean 13.4 +/− 4.4 cm).

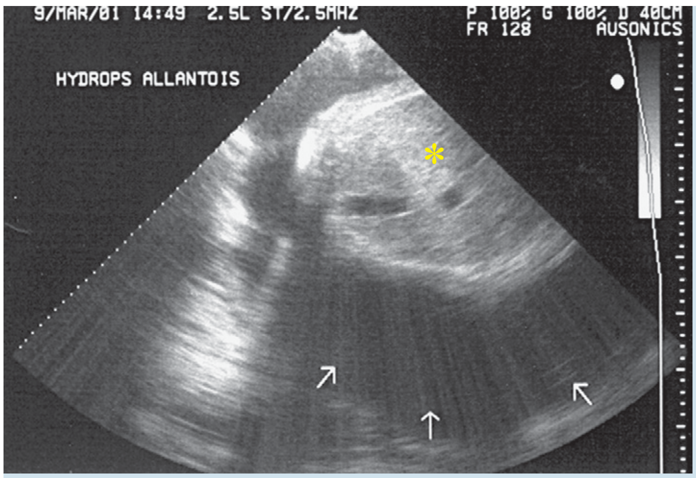

Figure 1.23: Transabdominal ultrasonographic image performed on a mare with hydrallantois during late gestation. The large amount of allantoic fluid (arrows) and the fetus (*) floating in it can be seen.

Figure 1.24: Mare with hydrallantois, the enlarged abdomen and distention of the ventral abdomen can be appreciated.

Figure 1.25: A mare with hydrallantois placed in stocks prior to draining the allantoic fluid. Administration of intravenous polyionic fluids should be part of the treatment – the objective of fluid therapy is to avoid the occurrence of hypotensive shock while draining the allantoic fluid.

- Rectal examination will reveal a large fluid-filled uterus with the dorsal wall protruding above the level of the pubis and absence of fetal ballottement.
- Transabdominal ultrasonography is the diagnostic tool of choice for confirmation of hydrallantois. The normal amount allantoic fluid depth is 4.7–22.1 cm with a mean maximal depth of 13.4 ± 4.4 cm. Thus *allantoic fluid depths of >18 cm are highly suggestive of hydrallantois.* An excessive amount of fluid is imaged from the ventral abdomen. Imaging of the non-pregnant horn via transabdominal ultrasonography usually reveals an abnormal amount of allantoic fluid.
- Edema of the ventral abdominal wall is usually present.

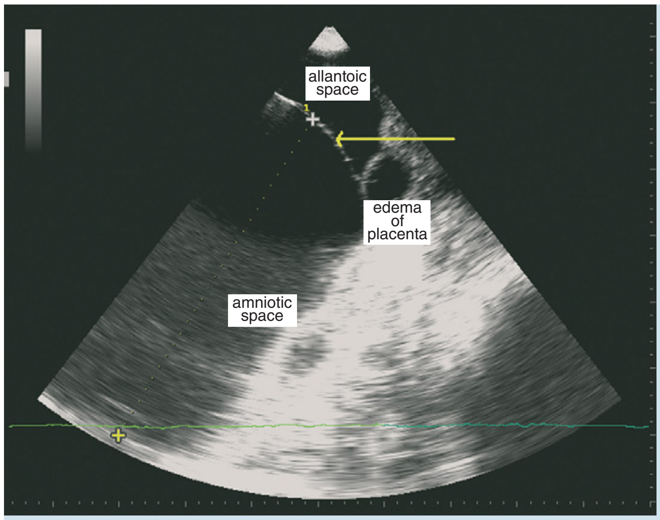

Figure 1.26: Hydramnion from a mare that had severe lymphedema of the placenta. The fetus could not be palpated during the rectal examination and unlike a hydrops allantois the uterus was not protruding above the level of the pubis. Normal fluid depth of the amniotic cavity is 0.8–14.9 cm (mean 7.9 +/– 3.5 cm). This mare's amniotic space measured 27.9 cm. The mare aborted the fetus at day 310 of gestation. Note the amnion (arrow).

Treatment

- The treatment of choice is early termination of pregnancy.
- The mare should be sedated and placed in stocks for easy management. The tail should be wrapped and the perineal area should be surgically prepared. Sterile technique should be followed during the procedure.
- Placement of an indwelling intravenous catheter is important for administration of polyionic crystalloid fluids while the allantoic space is slowly being drained; fluid therapy is advisable to avoid the possibility of hypotensive shock.
- Prostaglandin E (PGE) (available in gel form) can be used to dilate the cervix. After the cervix is gently dilated manually, the chorioallantoic membrane is located by palpation. The authors use a 28 French thoracic trocar to penetrate the membrane. The allantoic fluid is then drained in a slow and controlled manner.
- The abdominal musculature may be weakened by the stretching, and abdominal press during stage II of parturition may be compromised. Malpositioning and malpostures are common. Vaginal assisted delivery is recommended; however care should be taken not to traumatize the cervix.
- Retention of fetal membranes should be expected and appropriate treatment for removal and prevention of metritis–laminitis complex is indicated (see p. 27).
- Another complication is prepubic tendon rupture or ventral abdominal muscle rupture.

Ventral body wall hernias and prepubic tendon rupture (Figs 1.27–1.32)

Rupture of the transverse abdominus, rectus abdominus and oblique muscles and the prepubic tendon will result in alteration of the ventral abdominal sling of the pregnant mare. Muscle rupture of the

ventrolateral abdominal wall is more common than complete prepubic tendon rupture.

History

- Some breeds (draft) and older mares in addition to mares with hydrops of the fetal fluids are predisposed to the condition but in many cases the cause is not apparent.
- Most body wall defects and ruptures occur close to term.
- Body wall rupture will usually be preceded by marked ventral edema.

Clinical signs

- Marked ventral edema.
- Complete prepubic tendon rupture results in tilting of the pelvis such that the tail head and tuber ischii are elevated and a lordosis will be present. Cranial and ventral displacement of the mammary

Figure 1.27: A mare with rupture of the prepubic tendon. It is important to differentiate this condition from hydrallantois. Prepubic tendon rupture can also occur secondary to hydrallantois. Also note the elevation of the tailhead in this image.

Figures 1.28 & 1.29: Rupture of the ventrolateral abdominal wall in a mare during late pregnancy. Note the extensive edema. This mare went on to deliver a normal foal with assistance and is imaged again 6 months later in Fig 1.29. The edema is no longer evident but there is an apparent "dropping" of the ventral abdominal wall.

Figure 1.30: Localized rupture of the muscles of the lateral body wall.

Figure 1.31: Mare with a large ventral body wall hernia (courtesy of IEC).

Figure 1.32: Large amount of ventral edema in late pregnancy.

gland occurs as a result of the loss of the caudal attachment to the pelvis.

- Extreme pain associated with progressive enlargement of a ventral rupture may result in tachycardia.
- Rupture of the abdominal musculature may lead to incarceration of a segment of intestine and associated clinical signs.

Diagnosis and differentials

Definitive diagnosis may be difficult as ventral edema may arise from a number of other causes.

- The compressive weight of the gravid uterus on the venous and lymphatic drainage frequently results in marked edema of the ventral abdomen of the late gestation mare.
- Monitoring muscle enzymes (CK and AST) can help determine the severity of the condition. Increases indicate compromise of the musculature from the compressive weight of the gravid uterus.
- External trauma may cause large hematomas and significant ventral edema.
- Unilateral edema may be associated with external trauma, ventrolateral body wall damage or partial damage to the prepubic tendon. The presence of a hemorrhagic secretion in the mammary gland supports a diagnosis of tissue trauma.
- Ultrasonography may be useful in detecting the presence of incarcerated bowel and hematomas.

Treatment

- As a definitive diagnosis is often difficult to achieve, any mare showing marked ventral edema should be confined to a stall.
- Treatment options are largely dependent on the stage of the pregnancy, the anticipated value of the foal, the age of the mare and the suspected extent of damage to the muscles and prepubic tendon.
- Further tissue damage is likely as the mare approaches parturition – termination of the pregnancy may be the most humane option in some cases.
- However, as this is a condition seen in older mares the present foal may well be her last and a decision may be made to support the mare to term with the objective of obtaining a viable foal. If the foal is viable and mature, inducing parturition should be considered to prevent further enlargement of the defect.
- Belly bandages have been used as external abdominal support by transferring weight to the vertebral column. Stretching of the elasticated material of the belly band may decrease its effectiveness over time. Additionally, care should be taken to avoid pressure sores and regular replacement of padding is essential.
- An assisted delivery will be required if the mare carries to term or is induced as she will be unable to exert sufficient abdominal pressure to expel the foal.
- A caesarian section should be considered if incarceration of bowel is suspected or if the wellbeing of the foal is of primary concern and the prognosis for the mare is poor (as in most cases of prepubic tendon rupture).
- Abdominal wall defects are more clearly delineated as the edema starts to resolve following delivery. An abdominal support should be worn for 2–3 months, until the edema is completely resolved and a fibrous ring has formed at the site of the hernia.
- Successive surgical repairs of such hernias with polypropylene or plastic mesh have been reported and smaller hernias may heal by second intention. Repair of prepubic tendon ruptures is not possible.
- In cases where abdominal wall defects cannot be repaired and in cases of prepubic tendon rupture that survive delivery, re-breeding is not recommended. Embryo transfer is an option for certain breeds (where stud books allow) for owners who wish to continue to breed from such mares on the basis of superior bloodlines.

Uterine torsion (Figs 1.33–1.35)

Torsion of the uterus is uncommon but should be considered in any mare showing colic signs between month 8 of gestation and term. The degree of rotation can be from 180° to 540° in either direction.

Clinical signs

- Colic in the last trimester of pregnancy that is poorly responsive to analgesics.
- Dystocia in a term mare.

Diagnosis

- Rectal palpation is most commonly used for diagnosis. Usually a single tense broad ligament can be palpated as it passes dorsally from one side of the uterus to the other, indicating the direction of the torsion.
- Vaginal palpation is of limited value as, unlike cows, the torsion is usually cranial to the cervix.
- Transabdominal ultrasonography is useful if uterine rupture or fetal death is suspected.

Treatment

Various surgical and non-surgical techniques have been described. The technique chosen depends on the condition of the mare and fetus,

stage of gestation, value of the mare and fetus, and the duration of the condition.

Non-surgical

- De-rotation of the uterus in a term mare can be achieved by grasping the fetus ventrolaterally through a dilated cervix, and rocking the fetus back and forth until enough momentum is built up to then continue in an arc and thus rotate fetus and uterus back into a normal position. This is reported to be successful in 80% of cases of mares at term.
- Rolling the mare. This is more commonly selected in mares that are preterm. The anesthetized mare is placed in lateral recumbency on the side of the torsion. Ropes are applied to the limbs and a plank of wood with a moderately sized individual bearing weight on it is placed against the mare's flank in order to stabilize the uterus. The ropes are then used to roll the mare to the opposite side. A rectal palpation is then performed with the mare in sternal recumbency to determine if the procedure has been successful. The entire process can be repeated until de-rotation is achieved.
  - This procedure is controversial as there has not been a documented report of its safety.
  - Cited advantages are a low incidence of hemorrhage or recurrence and low economic cost.
  - Cited disadvantages are possibly making the condition worse if the direction of the torsion is misdiagnosed, the potential to displace the colon, and a high incidence of placental detachment.
  - There are varying reports as to the incidence of uterine rupture with the technique.

Figure 1.33: A uterine torsion has resulted in strangulation of the torso by the umbilical cord.

Surgical correction

- Standing flank laparotomy. This approach involves making an incision in the flank on the same side as the direction of the torsion. The surgeon then places a hand under the uterus and gently rocks back and forth until enough momentum is built up to de-rotate the uterus by a combination of a lifting and rolling movement. Occasionally a second incision in the opposite flank is required for a second surgeon to offer assistance by gently pulling over the top of the uterus. This procedure is more difficult in term mares with large fetuses or in cases in which the fetus is dead.
- Ventral midline celiotomy. This technique is used in cases where gastrointestinal involvement, uterine compromise or rupture is suspected. It also offers the advantages of allowing an emergency caesarian or ovariohysterectomy if required.
- The prognosis for the fetus depends on the degree of vascular compromise which in turn is dependent on the severity of the torsion and the speed of correction. However, in many cases correction is not timely enough and frequently uterine torsion

Figures 1.34 & 1.35: Correction of a uterine torsion by rolling.

is followed by abortion or the delivery of a dead foal in term mares.

- Where the fetus has been determined to be alive during correction in a term mare, preparations should be made for resuscitation and appropriate after-care as fetal hypoxia will make such foals likely candidates for hypoxic ischemic encephalopathy.
- In preterm mares that have been successfully de-rotated and the fetus has been determined to be alive, administration of progestins to suppress myometrial activity is recommended.

Infectious abortion

Equine herpes virus abortion (Figs 1.36–1.39)

History

Equine herpes virus 1 (EHV-1) causes abortion in mares during late gestation. EHV-1 can be responsible for isolated abortions or abortion storms; the former is nowadays a more common feature of the disease. The widespread use of vaccination combined with management practices has been responsible for the decreased incidence of abortions due to EHV-1.

EHV-1 can also be responsible for causing rhinopneumonitis. Clinical signs of respiratory disease such as fever, cough and serous nasal discharge are more common in younger horses. EHV-1 most commonly causes a subclinical respiratory infection in older horses, including brood mares.

Placental detachment occurs secondary to a virus-induced placental vasculitis. Infection of the fetus can occur via inhalation of amniotic fluid or via the chorionic vasculature.

Clinical signs

- Abortion occurs most commonly within 30 days post-infection, with the highest incidence in the last trimester.
- Clinical signs of disease are not observed in mares at the time of abortion.
- Fertility is not affected in mares that have aborted due to EHV-1 infection.
- Fetuses aborted in the last trimester due to EHV-1 are fresh. The most classical gross lesions seen in the fetuses are pulmonary edema, hydrothorax and necrotic foci in the liver and spleen.
- EHV-1 can also cause perinatal infection. In this case foals are born alive; however they are profoundly lymphopenic and generally die. The characteristic signs are weakness and respiratory distress; death occurs within the first 24–72 hours of life, despite aggressive therapy. (See Chapter 5 p. 147.)
- EHV-1 is also responsible for causing myeloencephalitis in all age groups.

Differential diagnosis

Abortions, especially during the last trimester of gestation, should be considered to be caused by EHV-1 until proven otherwise.

Diagnosis

- Clinical signs characterized by upper respiratory tract infection in younger horses and adult horses and late term abortions of fresh fetuses with the characteristic lesions are highly suggestive of EHV-1 infection.
- Examination of the aborted fetus reveals an absence of autolysis. The abdominal and thoracic cavities contain an increased amount of fluid. The liver is usually swollen and has necrotic white foci seen during gross examination.
- Tissue samples from the aborted fetus should be submitted to a diagnostic laboratory for confirmation of EHV-1. Samples of fetal lung, liver, thymus and adrenals should be submitted frozen and in formalin. Histological examination of the fetal lungs can show a hyperplastic, necrotizing bronchiolitis and the characteristic intranuclear eosinophilic inclusion bodies. Other characteristic histological findings are necrosis in lymphoid tissues, liver and adrenal glands.
- Neutralization tests, indirect immunofluorescence tests, PCR, fetal serology and virus isolation are some of the laboratory tests that can aid with the confirmation of the diagnosis.

Control

- There is no specific treatment for the EHV-1 induced abortion; therefore management practices are key factors for the prevention of abortion due to EHV-1.
- All pregnant mares should be vaccinated with a commercial vaccine at 5, 7 and 9 months of gestation, although vaccination does not absolutely protect against this problem and abortions may still occur. Others have recommended vaccinating pregnant mares every 2 months throughout gestation. All the different categories of horses at the farm should be vaccinated in order to decrease the shedding of the virus.
- Pregnant mares should be separated from weanlings and other horses and all new horses that arrive at the farm should be isolated for at least 3 weeks.
- The most dangerous source of infections for pregnant mares is an aborted fetus and the fetal fluids; therefore measures should be taken in order to prevent the contact of pregnant mares with aborted fetuses and fetal membranes. The area should be thoroughly disinfected with phenolic or iodinophoric compounds. All personnel in contact with the aborted fetus must change clothing and wash their hands before handling other mares.
- The farm should be quarantined until EHV-1 infection is eliminated as the cause of abortion.

Equine infectious anemia abortion

History

- Equine infectious anemia is caused by a retrovirus that is transmitted by horseflies, deerflies or iatrogenically with blood product transfusions or blood contaminated instruments. Aerosol spread has also been postulated during the 2006 outbreak in an Irish equine hospital.
- Mares infected with infectious anemia virus abort during the febrile stage of infection. Abortion can occur at any stage of gestation. It is likely that abortion is secondary to systemic illness.

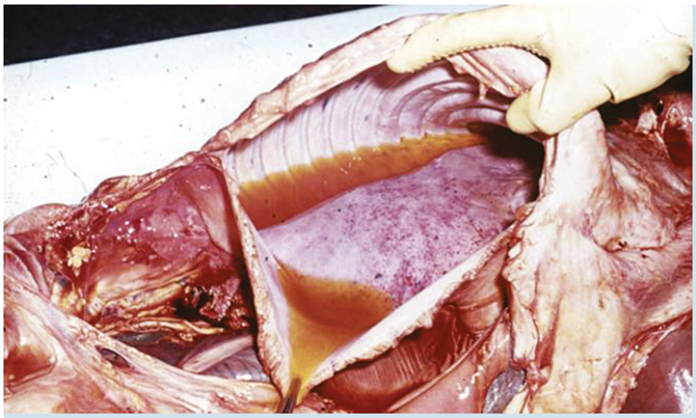

Figure 1.36: Hydrothorax in a late term EHV abortion (courtesy of IEC).

Figure 1.37: Pulmonary edema in a foal with EHV. Note the marked rib impressions on the lung surface (courtesy of IEC).

Differentials

Any condition that causes debilitation and anemia – however, abortion may occur in the early stages of disease thus confounding the diagnosis.

Diagnosis

Coggins test and C-ELISA are reliable in the diagnosis of equine infectious anemia.

Equine viral arteritis abortion

History

- Equine viral arteritis (EVA) is caused by a virus of the family Arteriviridae.
- Transmission can occur venereally from an infected or asymptomatic carrier stallion to a susceptible mare.
- Transmission via aerosols occurs for 21 days post-infection and it is this mode of transmission that is responsible for abortions and the rapid dissemination of disease through a susceptible population.

Figure 1.38: Necrotic foci in the spleen of a foal from a recent EHV abortion (courtesy of IEC).

Figure 1.39: Petechial hemorrhages on the oral mucous membranes of a 1-day-old foal that died with EHV infection (courtesy of IEC).

Clinical signs

- Systemic disease can vary from inapparent to severe. Signs include fever, leukopenia, conjunctivitis, nasal discharge, peripheral edema and vasculitis.
- Abortion is thought to be secondary to fetal anoxia due to placental edema and decreased progesterone production. Mares abort 7–10 days after the onset of acute clinical signs. Abortion usually occurs between the 5th and 10th month of gestation following infection

by the respiratory route. Abortions are uncommon in endemic populations.

Diagnosis

- EVA is a reportable disease in many countries.
- Virus isolation can be attempted from the buffy coat; however it can be difficult. PCR and immunofluorescent staining can aid in the identification of the virus from fetal and placental tissues.
- PCR or virus isolation can also be attempted from nasopharyngeal and conjunctival swabs, vaginal swabs and semen from an infected stallion.
- Autolysis of the aborted fetus is common because fetal death precedes abortion by a few days. Gross lesions found in the aborted fetus can include petechial hemorrhages on the pleural surface and mild pulmonary edema. Focal areas of hepatic necrosis or intranuclear inclusions are **not** a characteristic of this disease as they are in Herpes abortions.
- Serologic tests are used to demonstrate antibody titer. A four-fold increase in titer indicates recent infection. In cases of abortion a four-fold increase in titer may be difficult to demonstrate due to delay in taking the initial sample.

Differential diagnosis

Equine herpes virus 1, equine influenza and equine infectious anemia should be included in the differential diagnosis.

Control

- Vaccination of seronegative stallions with a commercial modified live virus vaccine where allowed.
- Breeding of carrier stallions should be limited to vaccinated or seropositive mares. Mares should be vaccinated annually, 3–4 weeks before being bred to a carrier stallion and these mares should be isolated for 3 weeks following breeding as they are capable of transiently shedding the virus following breeding.

Leptospirosis abortion

History

- Leptospirosis has been incriminated as a sporadic cause of placentitis, abortion and premature birth in horses.
- Pregnant mares are considered to be incidental hosts that become infected after exposure to a maintenance host.

Clinical signs

- Mares can exhibit systemic signs characterized by fever, anorexia, depression and icterus, with abortion occurring 1–3 weeks after clinical signs. However with *Leptospira pomona* infection clinical signs of disease are rarely exhibited by the mare.
- Abortion usually occurs between the 7th month of gestation and term.
- The aborted fetus shows evidence of icterus and autolysis. The placenta is edematous with a necrotic chorion covered with a mucoid exudate.

Diagnosis

- Diagnosis is based on isolation of the spirochetes, immunofluorescent staining and serology.
- Mares that abort usually have a high antibody titer to *Leptospira* sp. A rising titer along with abortion is considered to be diagnostic. However, a single microscopic agglutination test (MAT) on fetal fluids or maternal serum with a high titer (1 : 6,400 to 1 : 819,200) is highly suggestive.
- Leptospira can be detected in the fetus by fluorescent antibody test, silver stain or dark field microscopy. The fetal kidney and liver are the organs most likely to yield positive results with fluorescent antibodies.

Treatment and control

- Because horses can shed spirochetes in the urine for up to 3 months, infected horses should be isolated and antibiotic therapy should be instituted. Also, mares that have aborted should be isolated and the stall disinfected. Antibiotics effective against Leptospira are penicillin, oxytetracycline and enrofloxacin.
- Attempts should be made to prevent contact between maintenance hosts and pregnant mares. Cattle in the same property should be vaccinated. No commercial vaccines exist in the United States for horses.

Salmonella abortion

Salmonella abortus equi has not been isolated as a cause of abortion in the United States since 1932. Nowadays the non-host-adapted *Salmonella typhimurium* causes most of the equine salmonella abortions. The bacteria can be transmitted through ingestion of feed or water contaminated with feces or genital discharges.

Other causes of abortion

Cord torsion (Figs 1.40–1.46)

- Normal foals have an umbilical cord length of 36–83 cm with a mean length of 55 cm. Diseased cords at the Lexington Diagnostic Laboratory varied from 62–125 cm with an average length of 96 cm.
- Some amount of twisting is present in normal umbilical cords and only in cases where the umbilical cord shows local swelling and discoloration should the diagnosis of fetal death due to vascular obstruction be made. In such cases the fetus can be expected to be slightly to moderately autolyzed due to fetal death before abortion.
- Excessively long umbilical cords have been associated with entrapment of the fetal extremities or fetal trunk. In these cases the cord can be wrapped around the fetal thorax, neck or head and become compressed.
- In cases where the umbilical cord is too short, stillbirth can be caused secondary to premature intrapartum rupture of the cord and fetal asphyxia.

Trauma (Figs 1.47–1.50)

External trauma is a rare cause of abortion. The presence of the fetal fluids provides a buffer against external forces and thus such forces

Figures 1.40 & 1.41: Fresh abortion with twisting, edema and localized hemorrhage of the umbilical cord. This case also had an ascending placentitis. More than 12 twists of differing sizes are regarded as significant. Other evidence that the observed twisting is the cause of the abortion is areas of hemorrhage or vascular constriction along the cord.

Figure 1.44: Torsion of the umbilical cord, note the paler areas consistent with vascular constriction. The cord has been untwisted for this photograph (courtesy of IEC).

Figure 1.42: Abortion with marked hemorrhage and edema of the cord. At the time this photograph was taken the cord had been partially untwisted.

Figure 1.43: Umbilical cord torsion – multiple twists of differing sizes.

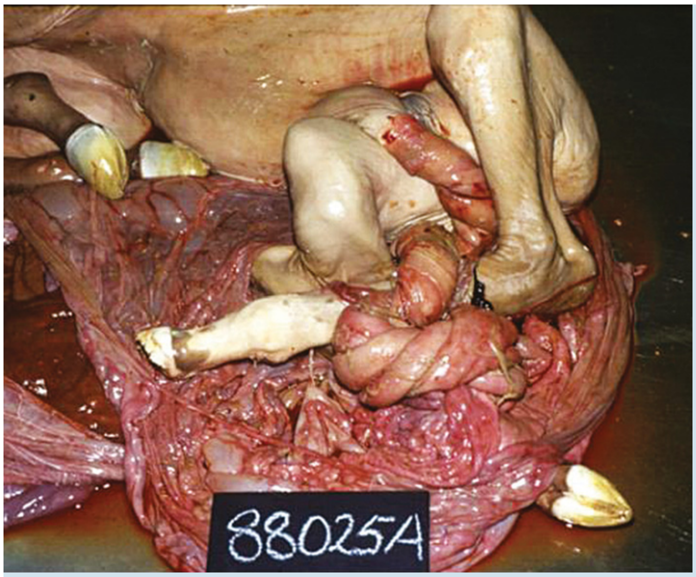

Figure 1.45: Umbilical cord wrapped around a hindleg (courtesy of IEC).

Figure 1.46: Umbilical cord wrapped around a hindleg.

Figures 1.47 & 1.48: Ventral abdominal wall hemorrhage and intra-abdominal hemorrhage in a recently aborted fetus. The mare was seen to have been violently kicked by another mare 8 hours prior to aborting and had extensive bruising of her lateral abdominal wall.

would have to be extreme in order to damage the fetus, uterus or uteroplacental unit.

Fetal abnormalities (Figs 1.51–1.54)

Most abnormalities of the fetus will result in early embryonic death but some may be carried closer to term. Fetal hydrocephalus is one such abnormality that may result in late term abortion.

Twins

- Equine twin pregnancies occur as a result of double ovulations. Some breeds (Thoroughbred) have a higher incidence of double ovulations and therefore twin pregnancies.
- Transrectal ultrasonography is used to diagnose twin embryos from day 11 of gestation. The equine embryo is motile until day 16 after which implantation occurs. In the case of twin pregnancies implantation can be unicornual or bicornual.
- A repeat examination should be performed at approximately day 25 of gestation; at this stage the fetal heart beat can be imaged. This examination is important to diagnose twin pregnancies that were missed in an earlier examination and to assess embryo viability and growth. It is important to remember to reduce or abort twin pregnancies prior to the formation of the endometrial cups that occurs between days 33–35 of gestation.
- Embryonic vesicles must be differentiated from endometrial cysts. Motility of the embryo up to day 16 of gestation can help to


Figures 1.49 & 1.50: Trauma to the mare resulted in this intrauterine metatarsal fracture and subsequent abortion.

Figure 1.51: Hydrocephalus in an aborted fetus. Also note the prognathia.


Figures 1.52 & 1.53: Hydrocephalus in an aborted fetus at 6 months of gestation.

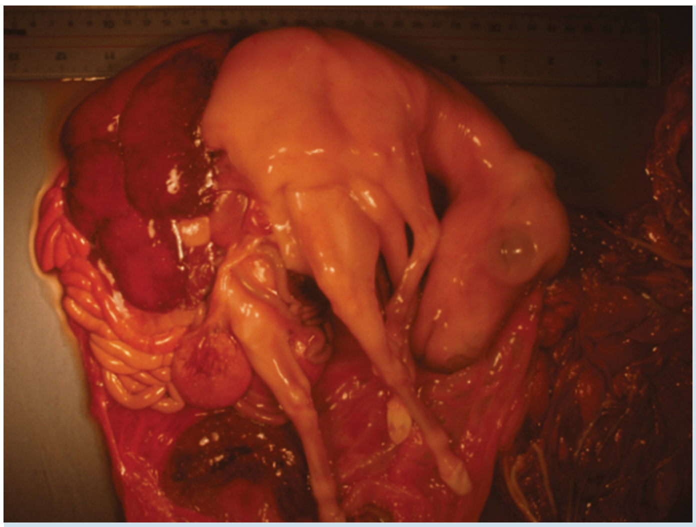

Figure 1.54: Schistosomus reflexus in an aborted fetus.

differentiate it from a cyst. Also, the increasing size of the embryonic vesicle (3–4 mm per day) aids in the differentiation.

- Transvaginal ultrasonographic guided allantocentesis before day 35 of gestation has been used as a means of reduction if earlier diagnosis or reduction was not possible. However, the success rate of this procedure has been low and in the authors' opinion should be avoided.
- Once the uterus descends into the caudal abdomen approximately at 120 days of gestation transabdominal ultrasonography is used for the diagnosis of twin pregnancies.
  - The late gestation gravid uterus extends along the ventral abdomen to the xiphoid.
  - By the 9th month of gestation the fetus should be in anterior presentation and dorsopubic or dorsolateral position. Thus in late gestation the fetal head should be positioned close to the pelvis of the mare.
  - Detection of a fetal head along the ventral midline indicates either an abnormal presentation or the presence of twins.
  - A complete examination of the uterus and fetus should always be performed when assessing fetal wellbeing and when determining if twins are present. Such an examination involves scanning in multiple parasagittal planes to the xiphoid and then scanning in multiple transverse planes, usually from left to right.
  - Identification of the non-gravid horn is helpful in ruling out the presence of twins.
  - Identification of two thoracic cavities and two hearts is essential when confirming the presence of twins.
  - An obvious size discrepancy may be detectable and in some cases where one fetus has already died a beating heart will not be detectable within the thoracic cavity.
- If twin pregnancy is detected later in gestation transabdominal ultrasound guided fetal cardiac puncture can be done between days 115–130 of gestation. The success rate of this procedure is variable and reported to be as high as 60% by some workers. Often-times however the surviving foal is small.
- A recent report describing craniocervical dislocation as a means of twin reduction between days 60–120 of gestation indicated a higher success rate than other techniques.
- In cases where transvaginal or transabdominal ultrasound guided approaches are used to terminate gestation, mares should be treated with non-steroidal anti-inflammatory drugs (flunixin meglumine), altrenogest and antibiotic therapy.

Fetal monitoring (Figs 1.55–1.66)

In human medicine, management of the late stages of pregnancy and the early neonatal period are greatly influenced by prepartum assessments of the fetus. In recent years many techniques have been used to assess equine fetal viability, of which ultrasonography of the fetus and uteroplacental unit has become widely used.

Transabdominal ultrasonography

- Correct preparation of the mare is important as it will improve the quality of the images obtained and reduce artifacts. The ventral abdomen should be clipped from the sternum to the udder either side of midline. Cleaning with a soapy solution and a final wipe of spirit will remove grease from the skin.
- The use of a biophysical profile (BPP) modified from that used in pregnant women is more efficient at predicting the outcome of a pregnancy than ultrasound scanning without grading of each parameter.
- The six parameters used are: FHR and FHR reactivity, fetal movement, uteroplacental unit (integrity and CTUP), fetal size, amniotic fluid volume and amniotic fluid quality.
- Where 0 represents abnormal and 2 represents normal, a score is applied to each parameter. A score of 10–12 indicates a positive outcome and a score <10 indicates a potentially negative outcome. However, events at parturition may result in the birth of abnormal foals despite normal profile values.

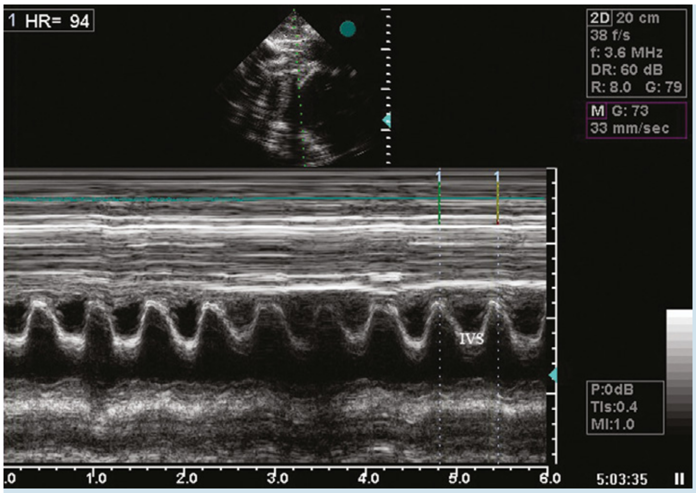

Figure 1.55: An M fetal echocardiogram with a corresponding two dimensional image mode image from the right ventral abdomen of a mare at approximately 9 months of gestation. The fetal heart rate can be calculated by determining the length of an individual cardiac cycle from the movement of the interventricular septum (IVS).

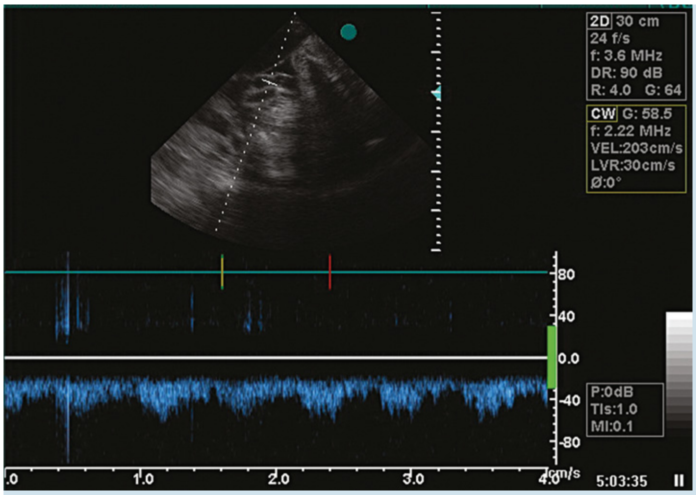

Figure 1.56: An M-mode fetal echocardiogram with a corresponding two-dimensional mode image from the right ventral abdomen of a mare at approximately 9 months of gestation. Fetal heart rate can also be calculated by determining the pulse through the umbilical vessels.

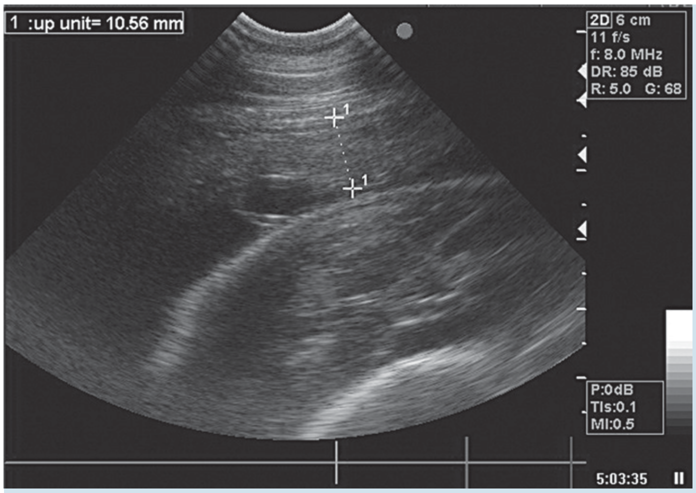

Figure 1.57: An ultrasonogram from the right ventral abdomen of a mare at approximately 9 months of gestation. The uteroplacental unit (between crosses) has a normal thickness (11.5 ± 2.4 mm).

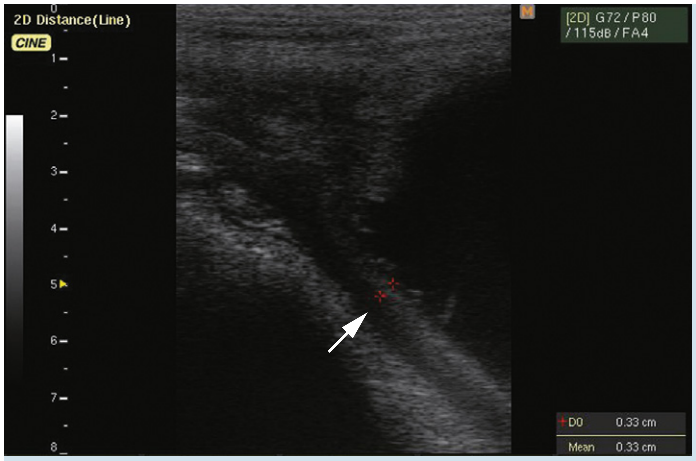

Figure 1.58: An ultrasonogram of a normal cervical uteroplacental unit. Note the anechoic area (arrow) under the measurement is not an area of separation but a uterine blood vessel.

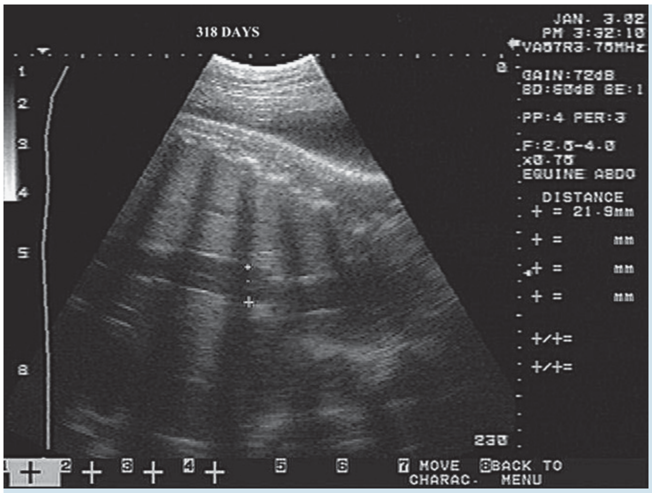

Figure 1.59: Aortic systolic diameter. The ultrasonogram shows a longitudinal view of the aorta. The image was obtained from a normal Thoroughbred mare at 318 days of gestation. The image should be frozen during systole and as close to the heart as possible. The calipers show the place where the aortic diameter was measured. In this mare the diameter of the aorta was 21.9 mm. The fetal heart is on the right of the picture.

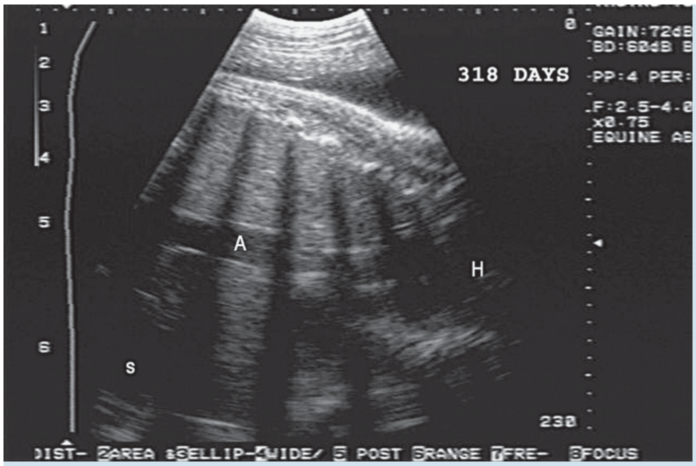

Figure 1.60: Aortic systolic diameter. The ultrasonogram shows a longitudinal view of the aorta. The image was obtained from a normal Thoroughbred mare at 318 days of gestation. The image should be frozen during systole and as close to the heart as possible. The normal values for fetal aortic diameter, measured in an average size Thoroughbred mare, vary between 6 mm at 140 days of gestation to 25 mm at 340 days of gestation. The main advantage of aortic diameter is that it is easy to measure throughout gestation and it has been strongly correlated with fetal size and outcome. A = aorta, H = heart, S = stomach.

FHR and FHR reactivity

- FHR is related to gestational age and fetal activity. FHR is normally regular and decreases from greater than 160 BPM before day 160 of gestation to 60–90 BPM in late gestation.
- FHR accelerations (+25–40 BPM) associated with fetal movement are regarded as normal.
- Persistent bradycardia is associated with fetal distress. Severe tachycardia and arrhythmias are associated with impending fetal death.

Fetal movement

- Fetal movement increases with increasing gestational age and periods of inactivity greater than 15 minutes warrant further evaluation.
- Ballottement, sound stimulation and oxytocin contraction tests can be used as fetal arousal techniques. Failure to respond indicates abnormality.
- Increased reflex activity occurs in the 3 days prior to foaling which allows the foal to obtain the correct position, presentation and posture for parturition.
  - *Position* refers to the relationship of the fetal spine to the dam's spine. Dorsal means the foal's spine is beside the dam's spine. Ventral means the foal's spine is away from the dam's spine.
  - *Presentation* refers to the area of the foal that is presented to the pelvic canal. Anterior means that the front of the foal is facing the pelvic canal, i.e. the foal is coming head first.
  - *Posture* refers to the placement of head and limbs relative to the body, i.e. whether the limbs are flexed or extended.

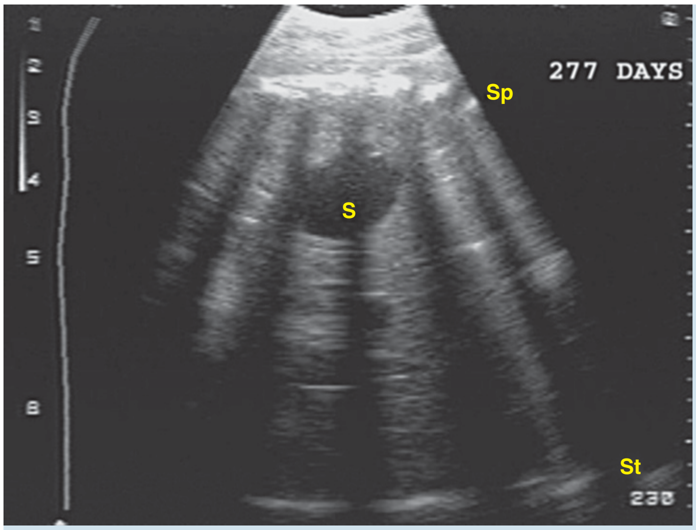

Figure 1.61: Thoracic diameter. The ultrasonogram shows a longitudinal view of a fetal thorax. The image was obtained from a normal Thoroughbred mare at 277 days of gestation. The landmarks for measuring the thoracic diameter are as follows:

1. Fetal spine (Sp), visualized as bright echogenic structures (vertebral bodies) with the rib shadows coming down from each vertebral body.
2. Fetal stomach (S), imaged as an anechoic rounded structure.
3. Fetal sternum (St), visualized as mixed echogenic structures.

The normal values for fetal thoracic diameter, measured in an average size Thoroughbred mare, vary between 80 mm at 140 days of gestation to 210 mm at 320 days of gestation. As with aortic diameter, fetal thoracic diameter has also been correlated with fetal size and outcome.

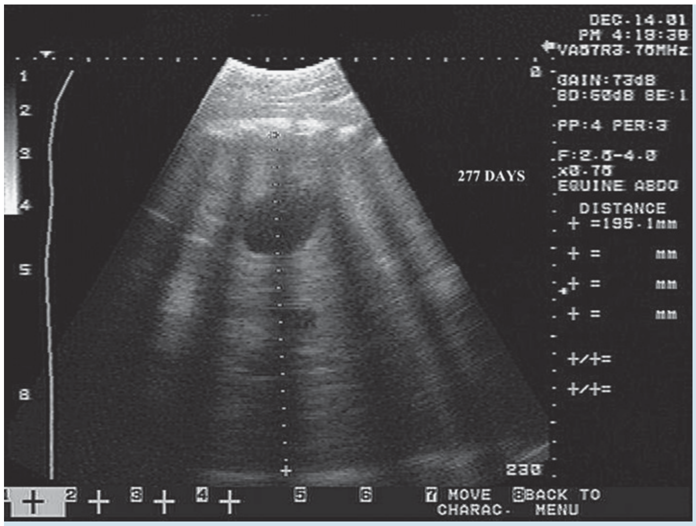

Figure 1.62: Fetal thoracic diameter measured between calipers – the measurement of this fetus at 277 days of gestation was 195.1 mm. The fetal thorax is an important area to visualize because several parameters can be determined, such as:

- fetal heart rate and rhythm
- fetal aortic diameter
- fetal presentation (should be cranial at the 9th month of gestation) and fetal position
- number of fetuses.

Fetal thoracic diameter is difficult to image late in gestation (after approximately day 310 of gestation) because the size of the fetal thorax will exceed the capacity of most ultrasound equipment, especially those used by most field practitioners.

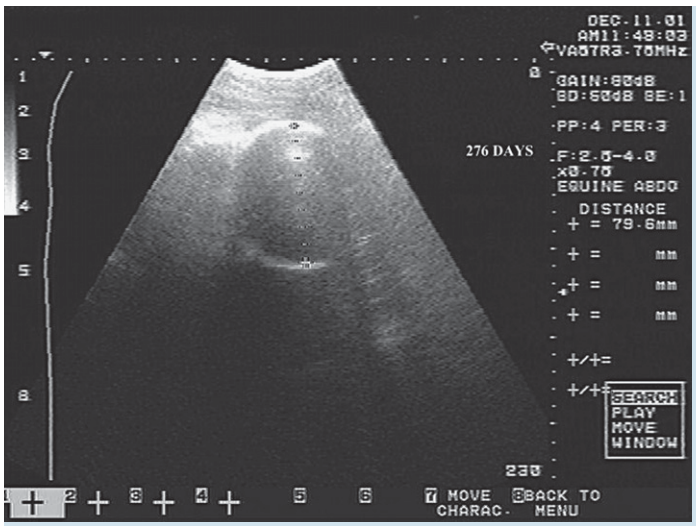

Figure 1.63: This figure depicts the frozen image of the fetal biparietal diameter of a normal equine fetus; when the maximal cross-sectional area of the skull was obtained the image was then frozen. The caliper cursors indicate the way to obtain the biparietal diameter: the caliper nearest the transducer should be placed in the inner margin of the skull and the caliper furthest away from the transducer should be placed in the opposite inner margin of the skull. The image was obtained from a normal Thoroughbred mare at 276 days of gestation.

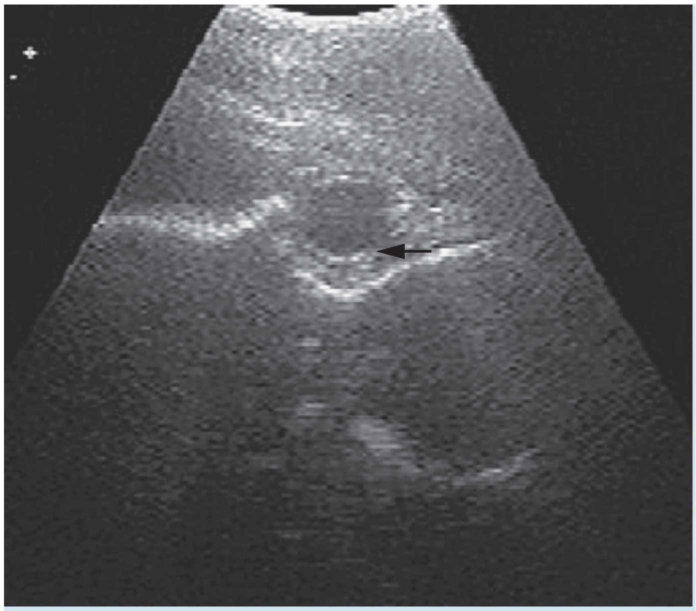

Figure 1.64: Eye volume. An image of a fetal eye, measured via transabdominal ultrasonography in a normal Thoroughbred mare at 277 days of gestation. Once the maximal cross-sectional area of the fetal eye was obtained the image was frozen. For eye depth, the calipers were positioned from the margin of the anterior portion of the eye to the area where the optic disc would be located (the optic disc itself is not imaged). The eye width was measured with calipers placed in a perpendicular line to the depth, where the maximal width of the vitreous body was seen. The following formula can be utilized to calculate the approximate fetal eye volume:

$$\text{Eye volume} = \text{Eye width} \times \text{Eye width} \times \text{Eye length}$$

Fetal size or fetal age does not have a good correlation with the fetal eye volume. A bright hyperechoic horizontal line can be seen in the center of the eye (arrow), this line represents the fetal lens. The lens is not always imaged in the normal fetal eye. Localization of the eye can also be of use in assessing the fetal presentation. During late gestation it can be difficult to image the fetal head.

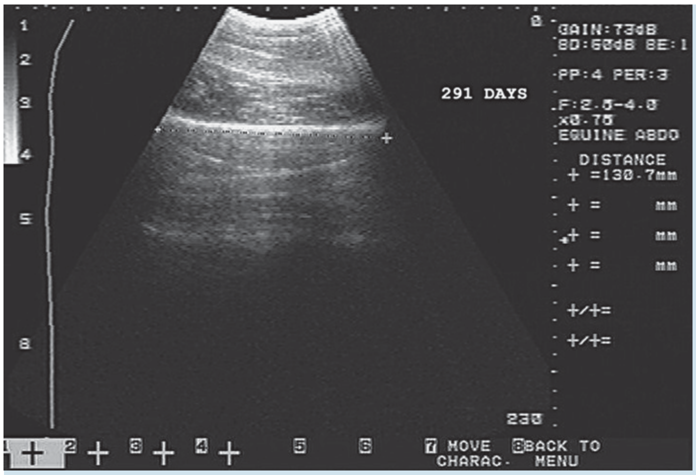

Figure 1.65: A longitudinal view of an equine femur at 291 days of gestation. The femur length in this fetus is 130.7 mm. The femur should be measured when its longest portion (diaphysis) is imaged (courtesy of Compendium on Continuing Education 2003 Fetal growth and development. 25(6):470–475).

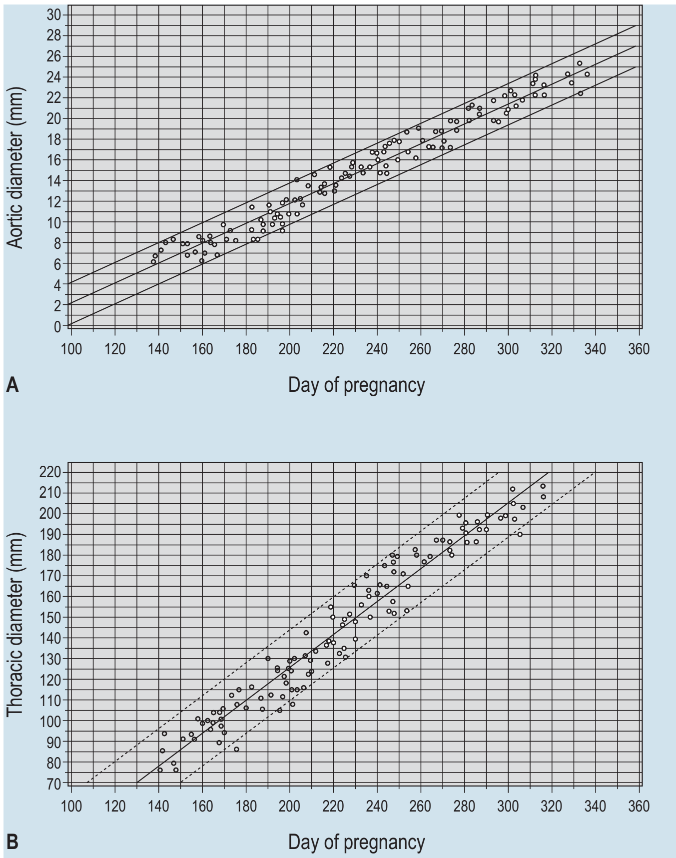

Figure 1.66: Graphs based on data from 33 normal pregnant Thoroughbred mares via transabdominal ultrasonography from day 130 of gestation until delivery. There is a linear relationship between aortic (A) and thoracic (B) diameters and days of gestation. See Figures 1.58–1.61 for how to measure aortic and thoracic diameter. (Courtesy of Compendium on Continuing Education 2003 Fetal growth and development. 25(6):470–475.)

Uteroplacental unit

- The uteroplacental unit should be assessed by both transabdominal and transrectal ultrasonography.
- Increases in the CTUP may indicate placental edema, placental separation or placentitis (see Figs 1.15–1.20).
- It is important to note that a large area of the uteroplacental unit cannot be visualized and that changes may occur quickly. In addition, placental abnormalities sufficient to cause decreased function may be subtle and not readily detectable on ultrasound examination.

Fetal size

- Fetal size assessments have typically been based on assessment of fetal aorta size. This is the most common measurement used as it is the easiest and most consistently accessible.
- A recent study has looked at using other measurements which appear to have a linear relationship with growth. Obtaining more than one measurement where possible is ideal.
- Other measurements which can be used are as follows:
  - biparietal diameter
  - eye volume
  - thoracic diameter
  - femur length.

The measurements taken can then be compared with growth charts allowing earlier diagnosis of intrauterine growth retardation, thus facilitating appropriate neonatal management.

Assessment of fetal fluids

- Both the allantoic fluid and amniotic fluid compartments can be imaged.
- Normal allantoic fluid depth is 4.7–22.1 cm with a mean maximal depth of 13.4 ± 4.4 cm
- Normal amniotic fluid depth is 0.8–14.9 cm with a mean maximal depth of 7.9 ± 3.5 cm. Amniotic fluid is normally more anechoic (clearer) than allantoic fluid with significantly fewer echogenic particles.
- The turbidity of fetal fluids increases with gestational age, however sudden increases or a marked turbidity of fluids may be associated with meconium passage, hemorrhage or inflammatory debris.

Predicting parturition

Measurement of colostral electrolytes can be used to indicate fetal maturity and predict impending parturition. Assessment of fetal maturity is important in determining timing of elective C-sections or timing of induction of parturition.

Calcium concentration rises sharply in the 24–40 hours prior to parturition (>40 mg/dL). Sodium concentration is typically higher than potassium concentration until 3–5 days before foaling at which time the ratio inverts; K > 30 mEq/mL and Na < 30 mEq/mL indicates fetal maturity in the normal pregnancy. Precise measurements are best performed with a laboratory chemistry analyzer.

Stallside test kits are available that measure $CaCO_3$, levels of 300–500 ppm indicate impending parturition within 12–18 hours.

Recommended reading

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CHAPTER 2

The post-foaling mare

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Introduction

Abnormalities in the neonate may be caused by events that occur in utero or events that occur at the time of parturition. A difficult delivery will commonly result in both neonate and mare requiring veterinary care. In addition to anticipated problems following dystocia, serious complications for the mare can also arise from apparently normal deliveries. This chapter reviews some of these postpartum conditions of mares.

Prolapsed uterus (eversion) and partial inversion (intussusception) of the uterine horn (Figs 2.1& 2.2)

History

- Intussusception of the uterine horn occurs when in the process of uterine involution the ovary and tip of the horn become inverted and trapped within the uterine lumen by a ring of myometrial spasm. Uterine inversion can progress to a complete uterine prolapse.
- Uterine prolapse occurs when the previously gravid uterine horn becomes invaginated after delivery and protrudes from the vulva. In mares uterine prolapse is uncommon, but it can occur secondary to normal foaling, retained placenta or dystocia.
- Uterine prolapse can be complicated by bladder eversion or prolapse, uterine rupture, intestinal herniation or uterine artery rupture.

Clinical signs

- With uterine horn inversion there is severe pain of acute onset within a few hours of foaling that does not respond to analgesic drugs.
- It is important to palpate the tips of both uterine horns per rectum when examining a post-foaling colic. The affected horn is shorter and extremely thickened.
- With uterine prolapse signs of shock are evident and it should be treated as an emergency.

Differential diagnosis

Uterine horn inversion and uterine prolapse should be differentiated from other causes of abdominal pain post-foaling such as:

- uterine contractions
- uterine hemorrhage
- gastrointestinal trauma – signs are usually seen 24–48 hours post-partum and are commonly associated with small colon trauma
- peritonitis secondary to gut trauma or damage
- peritonitis secondary to uterine tearing – signs are usually seen 24–48 hours post-partum
- colon torsion
- other causes of gastrointestinal obstruction.

Diagnosis

- In cases of uterine horn inversion the diagnosis is confirmed by examination of the uterus and ovaries via rectal palpation.
- In the case of uterine prolapse the uterus can be seen protruding through the vagina.

Treatment

Uterine intussusception

- If uterine horn intussusception is confirmed the mare should be sedated and in order to inhibit the uterine contractions, epidural anesthesia can be administered.

Figure 2.1: Uterine prolapse.

Figure 2.2: Replacement of a prolapsed uterus.

- Manual reduction is required with a hand placed in the uterine lumen. The ovary is grasped through the uterine wall and the fist is applied to the area of resistance until it gives way and allows the ovary and tip to be reduced.
- If standing reduction is not possible, reduction under general anesthesia is necessary.
- After the inversion is corrected the uterus should be lavaged with warm sterile saline solution in order to stimulate involution and reduce uterine contamination.

Uterine prolapse

- Uterine prolapse must be treated as an emergency.
- An indwelling intravenous catheter should be placed in order to administer fluid therapy and correct any electrolyte deficits, particularly calcium.
- Correction of the prolapse is aided by sedation and epidural anesthesia.
- General anesthesia is used in mares that exhibit intractable pain or excessive straining.
- The prolapsed uterus should be lavaged with hypertonic saline solution with or without glycerine or a hypertonic sugar solution to reduce edema, thus facilitating reduction.
- If uterine lacerations are identified absorbable suture should be used to close them. The uterus should be lubricated and gently replaced back through the vagina beginning with the uterine body and replacing the tip of the horns last.
- The uterus should be distended with sterile saline to ensure that the tips of the horns are extended fully.
- It is important to confirm that the uterus is placed in its normal position within the abdomen in order to prevent recurrence.
- Treatment should continue with broad-spectrum antibiotics, anti-inflammatory and fluid therapy.
- Tetanus prophylaxis is also recommended.
- Local intrauterine therapy such as lavage and antibiotic infusion is recommended; however this will depend on the condition of the uterus. Close monitoring is warranted due to the risk of developing metritis, laminitis or septicemia.
- The combination of shock, hemorrhage, contamination and uterine trauma warrants a poor prognosis in cases in which treatment is delayed. Cases which are seen early and promptly treated may make a full recovery with no adverse effects on future fertility.

Prolapsed bladder and prolapsed viscera (Figs 2.3 & 2.4)

- The bladder or bowel can prolapse with the uterus. Ultrasonography of the prolapsed uterus can be useful in the evaluation of any suspicious contents.
- If a prolapsed bladder is confirmed, then aspiration of its contents should be performed through a large bore needle. In the case of a loop of bowel a ventral midline celiotomy may be required for correction and evaluation of gut viability.
- Bladder and bowel prolapse can occur independently of uterine prolapse. Bladder prolapse is associated with excessive straining at the time of foaling. Bowel prolapse can occur secondary to uterine or rectal tears. Prognosis is good in cases of bladder prolapse in which the condition is recognized and corrected quickly. However in most cases of bowel prolapse the prognosis is poor.

Uterine tears (Figs 2.5–2.7)

History

- Uterine tears can occur secondary to dystocia or during apparently normal deliveries.

Figure 2.3: Prolapsed urethra post-foaling.

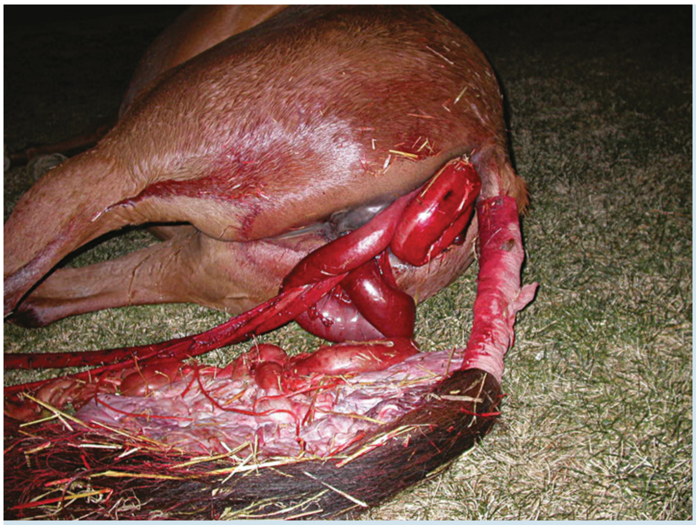
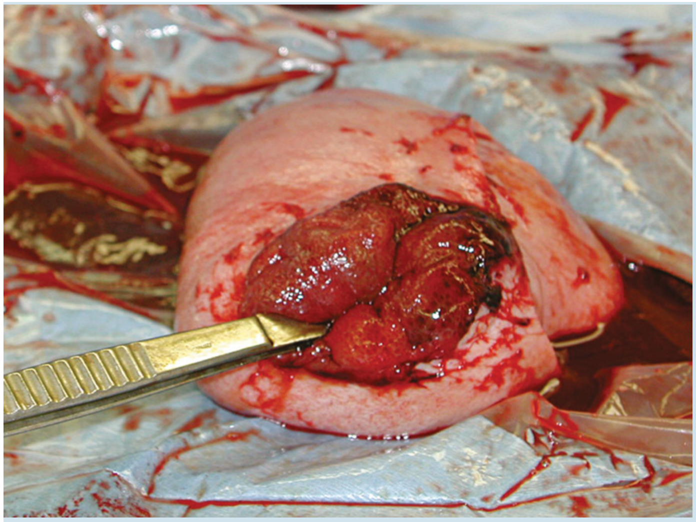
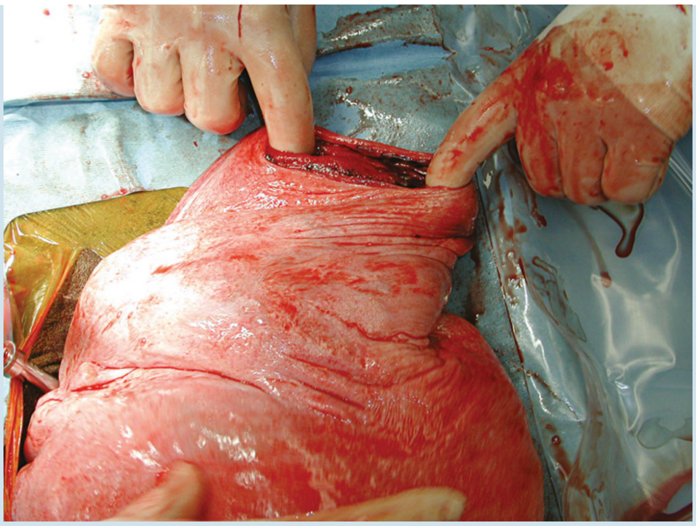
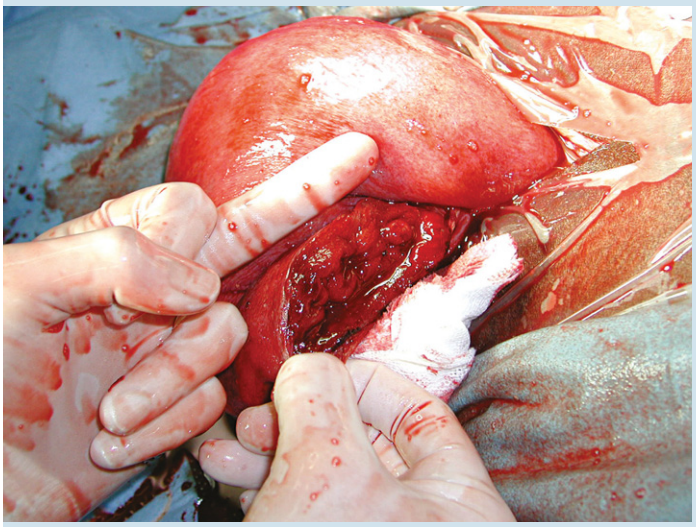


Figure 2.4: Prolapse of bowel post-foaling. Bowel prolapse may occur in association with uterine prolapse or may occur secondary to uterine or rectal tears.







Figures 2.5–2.7: Uterine tear at the tip of the gravid horn.

- Occasionally a hoof of the foal may be forced through the dorsal uterine wall during the expulsive efforts of the mare; in these cases a loop of bowel can protrude through the vulva.
- Another common place for a tear is in the ventral body at the pelvic brim.
- A common site of uterine tear in unassisted deliveries is the tip of the gravid horn.

Clinical signs

The clinical signs depend on the degree of the uterine tear. Affected mares show signs of abdominal pain, fever, lethargy and anorexia as peritonitis develops.

Differential diagnosis

Other causes of postpartum colic such as: uterine contractions, uterine hemorrhage, broad ligament hematoma and bowel damage with secondary peritonitis.

Diagnosis

- It is important to keep in mind that the interval between the occurrence of the tear to diagnosis and treatment has a significant effect on the prognosis and survival.
- Uterine palpation via the cervix should be done in an attempt to locate the uterine tear; however this is difficult in a postpartum mare as the tip of the uterine horns can be out of reach. Thus the inability to find a tear by this means does not preclude its presence.
- Abdominocentesis should be performed in order to determine the presence and degree of peritonitis. In cases of uterine tear the peritoneal fluid contains an elevated number of red blood cells, total proteins and white blood cells, with intracellular and extracellular bacteria sometimes seen on cytological examination.
- Ultrasonographic examination of the abdomen frequently reveals the presence of moderate to large volumes of free abdominal fluid. The uterus will often have a reduced amount of fluid as some has leaked into the abdomen.
- When uterine artery hemorrhage has been ruled out, it may be necessary to do an exploratory celiotomy to make a definitive diagnosis.
- Laparoscopic examination of the uterus may confirm the diagnosis and provides information to determine the need for surgical intervention; however, if this is not practical surgical intervention may be required before the patient deteriorates significantly.

Treatment

Supportive care includes systemic broad-spectrum antibiotic therapy, non-steroidal anti-inflammatory drugs, intravenous fluid therapy and peritoneal lavage. Oxytocin therapy can be used to aid with the uterine involution.

Most tears need a ventral celiotomy in order to be surgically repaired.

Uterine hemorrhage (Figs 2.8 & 2.9)

History

Hemorrhage from the uterine artery is common in older (>11 years) multiparous mares. In these mares hemorrhage from the uterine artery is a significant cause of periparturient colic and death.

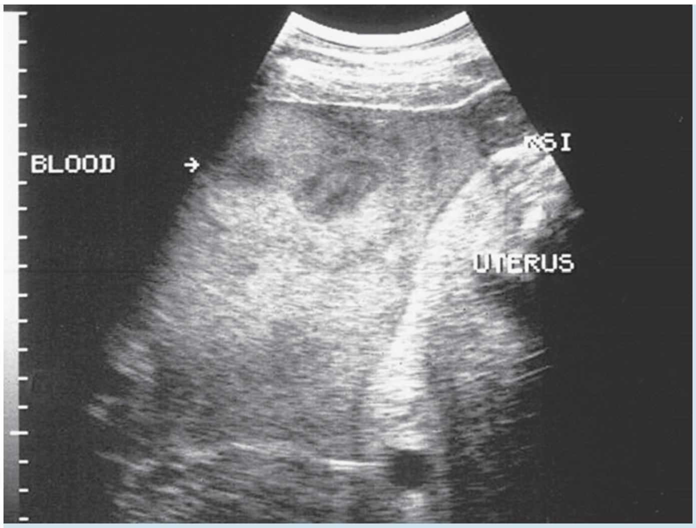


Figure 2.8: Ultrasonogram from the ventral abdomen of a 20-year-old mare with a uterine artery rupture demonstrating free blood in the abdomen. Blood is typically visualized as echoic fluid with a swirling pattern.

Figure 2.9: Broad ligament hematoma at post-mortem in a mare that suffered a uterine artery hemorrhage post-foaling.

Hemorrhage is not always fatal, as it may slowly dissect into the broad ligament between the myometrium and the serosa of the uterus, forming a hematoma.

Clinical signs

- If the bleeding is contained within the broad ligament signs of severe abdominal pain (anxiety, tachycardia, tachypnea, sweating) are seen.
- Examination of the mucous membranes reveals pallor.
- If the bleeding occurs into the abdominal cavity or if the broad ligament ruptures, then the mare shows signs of hemorrhagic shock or is found dead.

Differential diagnosis

Other causes of postpartum colic such as: uterine contractions, uterine tears and bowel damage with secondary peritonitis.

Diagnosis

- Clinical signs of colic and hemorrhagic shock.
- Transabdominal ultrasonography, abdominocentesis and hemogram are useful to confirm the diagnosis of hemorrhage. In evaluating the hemogram a decrease in total protein will be seen before a decrease in hematocrit.
- Hemorrhage usually occurs in mares >11 years old that have had more than one foal, however it has been reported in young maiden mares and consequently it should always be considered as a differential in any postpartum colic.

Treatment

- The treatment of mares with post-foaling hemorrhage varies widely among clinicians and referral institutions.
- Minimizing the level of stress on the mare is an important part of the treatment. If hemorrhage is suspected, some or all of the following methods of minimizing stress may be required:
  - place the mare in a quiet, dark stall
  - analgesic therapy is essential and sedation is required in many cases
  - minimize handling
  - avoid separation from the foal unless the foal is in grave danger.
- An indwelling intravenous catheter should be placed. Different types of intravenous fluids (polyionic crystalloids, colloids and/or hypertonic saline solution) and different administration protocols have been recommended.
- Antifibrinolytic drugs (aminocaproic acid) may aid with clot stabilization.
- Blood transfusion from a universal donor should be administered if the hematocrit drops below 15%.
- Intranasal oxygen at a volume of 10 L/min at a continuous flow rate can also be given.
- If the mare survives the hemorrhagic crisis then broad-spectrum antibiotics, anti-inflammatories, pain management, nutritional support and good nursing care are important factors in the treatment.

Retained fetal membranes (Figs 2.11–2.17)

History

- Fetal membranes should normally be expelled within 3 hours post-foaling. Placental retention is more likely to occur secondary to

Figure 2.10: Ruptured broad ligament hematoma. Note the site of rupture (arrow). This hematoma was known to be present following foaling. The mare progressively lost weight over the next month with intermittent colic signs. The mare was euthanized at 1 month post foaling when she developed signs of severe colic and hemorrhagic shock. Post mortem examination revealed a loop of small intestine (*) adherent to a large broad ligament hematoma and a tear within the capsule of the hematoma which had resulted in an intra-abdominal hemorrhage.

Figures 2.11: Retained fetal membranes.

Figures 2.12 & 2.13: Partial retention of the fetal membranes. Fig 2.12 shows the placenta as it was passed with the birth tear on the left hand side and a tear in the non-pregnant horn on the right hand side. The missing portion of the placenta was retained within the uterus. Lavage of the uterus resulted in retrieval of the missing segment imaged in Fig 2.13.

Figure 2.14: Ultrasonogram obtained from the right ventrolateral abdomen of a post-partum mare with metritis. The image shows edema of the uterine wall with a large volume of echoic exudates within the uterine lumen.

abortion, dystocia or cesarean section – in these cases tissue inflammation appears to be a predisposing factor.

- After any foaling the expelled placenta should be carefully examined to ensure that it is intact as small portions may be retained.
- Metritis and laminitis are common sequelae.
- In the authors' opinion when a mare shows fever and/or depression in the early postpartum period the uterus is the first place to look for a problem as retained fetal membranes and metritis are the most frequent causes.

Clinical signs

- Portions of the placenta can normally be seen protruding from the vulva; however, a portion of the placenta can be retained within the uterus.
- If retained placenta is uncomplicated then systemic signs are not present.
- In cases of toxic metritis the mare shows fever, lethargy, anorexia, tachycardia, tachypnea, injected mucous membranes and signs of laminitis. Palpation of the uterus per rectum reveals a large, fluid filled, thin walled and atonic uterus. The fluid within the uterus is fetid, thick and red to brown in color. Vaginal discharge may or may not be observed.

Differential diagnosis

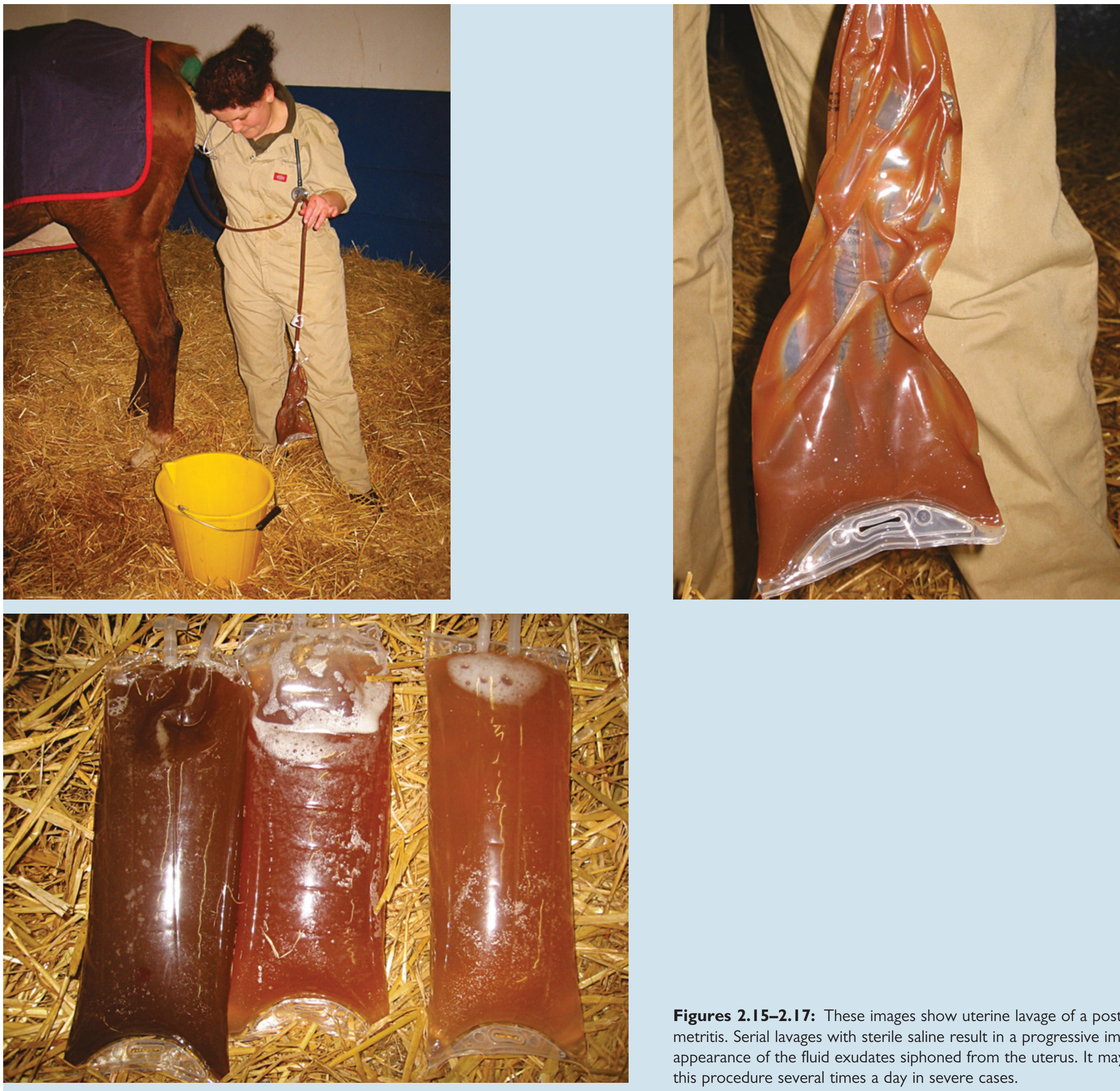
- Normal postpartum fluids.
- Depression in the postpartum period may also arise as a result of peritonitis caused by uterine tears or bowel damage.

Diagnosis

- The fetal membranes should be examined post-foaling in order to determine their integrity (see Figs 2.38–2.71).
- Clinical signs and examination of the uterus.
- Complete cell count usually reveals leukopenia and neutropenia.

Treatment

- Treatment will depend on the duration of membrane retention and presence or absence of metritis with septicemia.
- In uncomplicated cases of retained placenta a low dose of oxytocin (10–20 IU) should be administered IM initially. This should be performed if the membranes have not been passed at 3–4 hours post-foaling. Up to six doses should be given at 1-hour intervals.
- The sensitivity to oxytocin varies widely and a positive response is indicated by the passage of fluid from the vagina. If no signs of discomfort such as sweating and restlessness are noticed within 10–15 minutes of the IM injection then the dose of oxytocin can be



Figures 2.15–2.17: These images show uterine lavage of a post-partum mare with metritis. Serial lavages with sterile saline result in a progressive improvement in the appearance of the fluid exudates siphoned from the uterus. It may be necessary to repeat this procedure several times a day in severe cases.

increased in increments of 10–20 IU until a positive response is seen.

- ***In cases where the fetal membranes have been retained for 6–8 hours when one first examines the mare or the mare has already failed to respond to six doses of IM oxytocin, systemic antibiotic therapy is indicated. If the mare is being seen for the first time and has not already received oxytocin then IM oxytocin should be commenced in conjunction with antibiotic therapy.*** If the mare has already received IM oxytocin and failed to respond then an IV drip of oxytocin (100 IU of oxytocin/L) should be started. The drip rate will vary on the individual mare but signs of contractions every 10–15 minutes should be apparent.
- Uterine lavage should also be performed – two methods have been described:
  - The first involves infusion of fluid (1% povidone–iodine) through a wide bore stomach tube into the chorioallantois by finding the birth tear and advancing through it. Enough fluid is placed to just cause overflow. The tube is withdrawn and the membranes are tied. Oxytocin is then administered to cause contraction of the uterus against the distended membranes. If the procedure is unsuccessful it may be repeated a number of hours later.
  - The second method, which is more commonly used for partial retention of the membranes, is to infuse 1% povidone–iodine between the membranes and uterus and then immediately siphon it off. This can be repeated 2–3 times with small volumes (1–4 L) of fluid. If the section of membrane can be reached then it may be gently teased off the endometrium and removed. However, if it is firmly adhered then traction should be avoided as it may result in uterine inversion or tearing.
- Cases of toxic metritis should be treated with broad spectrum systemic antibiotics, anti-inflammatory drugs, anti-endotoxic drugs and intravenous fluid therapy. Tetanus prophylaxis is also recommended. The uterus should also be lavaged with large

volumes of sterile fluids several times a day. Intrauterine infusion with antibiotics is recommended by some authors but remains controversial.
- Prophylactic treatment for laminitis should include soft deep bedding, hoof pads and pain management.

Perineal laceration and rectovaginal fistulas (Figs 2.17–2.25)

History

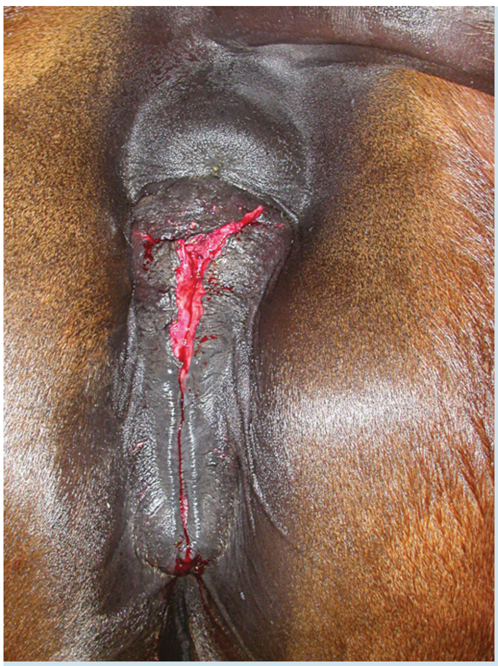
- Primiparous mares are more susceptible to injury of the reproductive tract and associated structures during parturition.
- Perineal lacerations can be classified by the extent and severity of tissue damage:
  - *First degree laceration*: involves the mucosa of the vestibule and skin of the dorsal commissure of the vulva.
  - *Second degree laceration*: involves the vestibular mucosa and submucosa, skin of the dorsal commissure of the vulva, and perineal body musculature including the constrictor vulvae.
  - *Third degree laceration*: involves the floor of the rectum and ceiling of the vestibulum, musculature of the perineal body, perineal septum and anal sphincter.
  - *Rectovaginal fistula*: involves the ceiling of the vestibule, floor of the rectum and a variable amount of the perineal septum and musculature. The fistula can be located close to the cutaneous perineum or deep into the vestibule.

Diagnosis

- The diagnosis is easily made by examining the external genitalia and vagina.
- Rectal palpation, ultrasonographic evaluation of the uterus and speculum examination of the vagina and vestibule should be performed in any mare with a foaling injury.
- Uterine biopsy may be recommended for older mares in order to identify and eliminate mares from having surgery that are unlikely to subsequently conceive.
- Other factors that must be considered before surgical correction include age of the mare, breeding history, whether artificial insemination is permitted in that breed, and whether surgery will offer only a short-term solution to the problem.

Treatment

- The prognosis for future fertility after successful surgical repair is good, with a conception rate reported to be as high as 75%.
- Surgical treatment varies according to the degree of injury. ***NO*** treatment should be attempted until the inflammation from the trauma of the injury has subsided.
  - *First degree lacerations* are surgically repaired with a Caslick procedure. Any trauma and inflammation must be allowed to subside before the Caslick procedure is performed. This must not be attempted when the area is inflamed and there is superficial infection as the sutures will not hold.
  - *Second degree lacerations* require a delayed surgical reconstruction of the perineal body as a Caslick procedure will not completely prevent vaginal contamination with air and/or feces.



Figures 2.18: First degree laceration showing tearing of the dorsal commissure of the vulva.

Figure 2.19: Third degree perineal laceration. Note the extensive bruising, absence of mucosa and retained fetal membranes in this case.

Figures 2.20 & 2.21: Third degree perineal laceration seen 12 hours after foaling. Extensive external swelling and bruising are evident. Closer examination reveals extensive fecal contamination.

Figure 2.22: This image shows a mare with a third degree perineal laceration prior to surgery. The inflammation and edema have subsided leaving a healthy bed of granulation tissue. Note the urinary catheter in place.

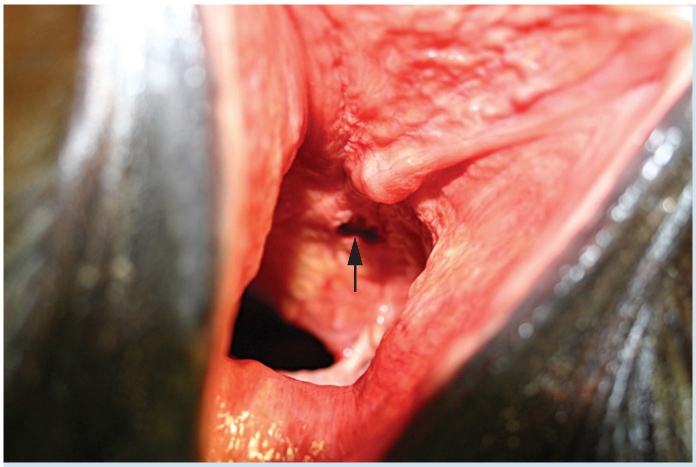


Figure 2.23: A small rectovaginal fistula prior to repair (arrow). This mare had a previous rectovaginal fistula repair and had subsequently conceived and delivered another foal. At foaling she developed a small fistula at the site of the previous repair. This fistula was not readily palpable after foaling and was suspected on the basis of recurrent uterine infections. A stained solution was placed in the rectum and an immediate speculum examination of the vagina revealed the site of the fistula.

- *Third degree lacerations* cannot be repaired during the acute period due to the excessive inflammation, edema and tissue necrosis. Thus attempts to surgically repair these lesions should be delayed for at least 3–6 weeks from the injury time and some more severe cases may require longer. Medical treatment during the acute period consists of administration of tetanus toxoid, systemic antibiotic therapy and systemic non-steroidal anti-inflammatory drugs. The wound should be cleaned daily. It is also important to change the mare's diet during the perioperative period with the objective to soften the fecal consistency at the time of surgery and during the postsurgical period.

Different surgical techniques have been described but all have the same goal, which is the re-establishment of a shelf between the rectum and vestibule and restoration of a functional perineal body.

Vulval hematoma (Figs 2.24–2.26)

History

Vulval hematomas occur most commonly in primiparous mares or in mares that have extremely large foals. Many breeders pay little attention to the foal size of particular stallions. We need to pay more attention to birth weight, as do the breeders of cattle, and some of these problems could be reduced.

Clinical signs

Unilateral vulval swelling.

Figures 2.24–2.26: Vulval hematomas.

Differential diagnosis

Edema caused by birth trauma or a vulval abscess as a result of mucosal tears in the vestibule.

Diagnosis

Palpation and/or ultrasonographic examination of the area.

Treatment

Except in extreme cases these conditions should be left alone as they will resolve on their own. In some instances the lancing of an abscess may hasten recovery, but unless hematomas are very large they will do better if left to resolve.

Vaginal hematoma / abscessation

History

Hematomas of the vagina are in most instances a result of dystocia and/or an extremely large foal. They are sometimes visible from the outside but in many cases are not found until the mare is examined for breeding

Clinical signs

Swelling in the vaginal vault is usually the only clinical sign. This can be visualized on speculum examination and palpated rectally.

Differential diagnosis

The primary differential is to differentiate between an abscess and a hematoma. A fractured pelvis could be a complicating factor but the mare would usually be showing other signs such as lameness.

Diagnosis

- Diagnosis is usually made on a speculum examination and/or a rectal palpation. A large mass is palpated in the pelvis.
- Ultrasonographic examination will often give a definitive diagnosis. In the author's opinion most abscesses start out as hematomas and then became infected.

Treatment

- In most instances these are best left alone. The hematomas will resolve in a matter of months and unless they are very large will have little effect on fertility.
- Abscesses can be more problematic and in some instances may need to be drained. Depending on the location of the abscess this can be done from inside the vagina or from the outside lateral to the vulva. Care must be taken when draining as there are many large vessels in this area.

Urinary incontinence (Figs 2.27 & 2.28)

History

Urinary incontinence occurs most frequently following the birth of a large foal or dystocia with accompanying swelling and bruising of the caudal reproductive tract.

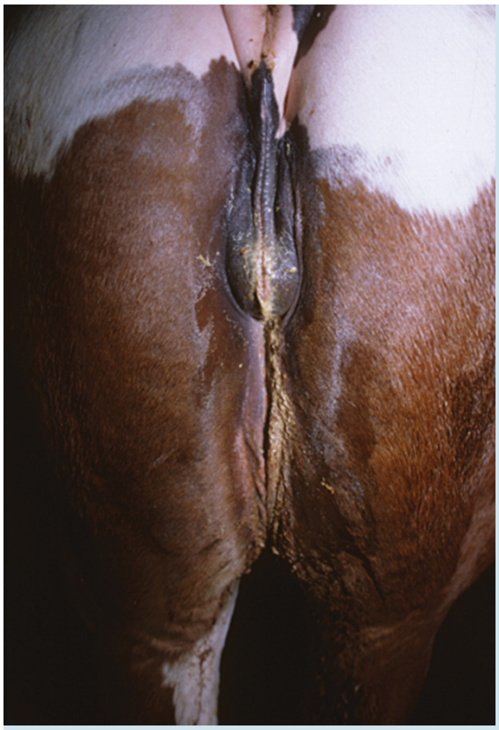


Figure 2.27: Urinary incontinence in a mare 2 months post-foaling.

Figure 2.28: Chronic urinary incontinence in a mare post-foaling. Note the extensive contact dermatitis.

Clinical signs

- Mare does not posture correctly to urinate.
- Urine is present on hind legs and tail.
- Mare does not actively urinate; urine appears to randomly splash out of vagina.

Differential diagnosis

- Vaginal urine pooling.
- Severe urethral irritation.

Diagnosis

Inability to posture and on rectal examination the urinary bladder is greatly distended and cannot contract. Urine pooling should be ruled out by speculum examination.

Treatment

Anti-inflammatory drugs and time. Placement of a urinary catheter in the acute stages will prevent bladder distension. Most of these mares will correct themselves when the inflammation subsides.

Miscellaneous gastrointestinal conditions (Figs 2.29–2.32)

The following conditions may all result in signs of moderate to severe colic in the days after foaling:

- Moderate to severe peritonitis may occur in the postpartum mare as a result of bruising of the small colon, cecum or rectum that occurs when the involved segment of intestine is trapped between the uterus and pelvis at foaling.
- A portion of mesentery may be torn from the intestine at foaling, resulting in ischemic necrosis of the affected segment of bowel.
- Segments of bowel may also become incarcerated in mesenteric rents that occur at foaling.
- Rupture of a viscus incites a massive peritonitis and septic shock. The condition is rapidly fatal and if recognized the mare should be euthanized. The tip of the cecum is the most likely site of rupture in the alimentary tract but other sites can be affected.

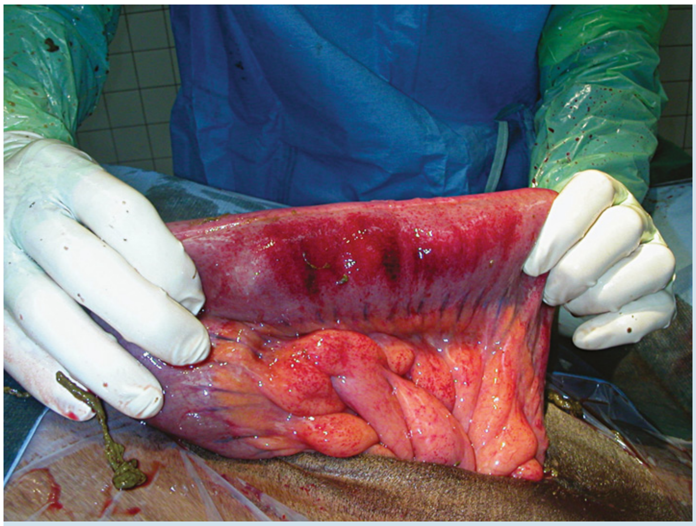
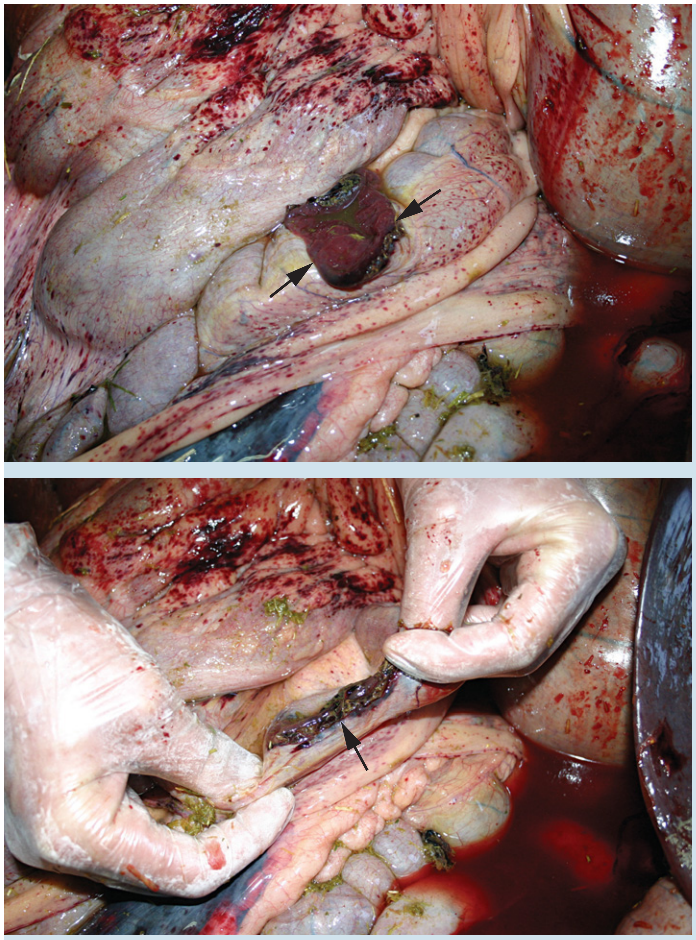


Figure 2.29: Extensive bruising and edema of the small colon post-foaling. The mare in this image also had extensive bruising of her stomach wall and ruptured her stomach 24 hours post-foaling.

Figure 2.30: Fecal matter present in the abdomen of a mare who ruptured her cecum pre-foaling.



Figures 2.31 & 2.32: Same mare as Fig 2.31. Note the area of cecal rupture (arrow) and the everted mucosa in Fig 2.32. Also note the extensive bruising of other areas of the bowel.

Mastitis (Figs 2.33–2.36)

Mastitis can occur at any stage of lactation and can in rare situations occur pre-foaling. It is most commonly seen within a few weeks of weaning or in situations where a foal is nursing poorly or there is an abrupt cessation of nursing such as the death of a foal.

Diagnosis and clinical signs

Diagnosis is based on clinical signs and microbial culture. Signs include:

- Swelling of the udder, which is usually hot and hard to touch. Severe cases may be accompanied by ventral edema extending as far forward as the sternum.
- Mammary secretions become thick and have a dark yellow appearance. Clots may be apparent in some cases. A sample should be obtained taking great care to avoid contamination. In severe cases it may be difficult to obtain any sample.
- *Streptococcus zooepidemicus* is the most common isolate but other bacteria, including Gram-negatives, can also be isolated so culture and sensitivity testing are important.

Figures 2.33 & 2.34: Enlargement of the mammary gland associated with mastitis. This mare's foal died suddenly at 5 months of age. The second image shows the thick, dark yellow mastitic milk sample.

Treatment

- Removing as much of the mastitic secretion as possible is an important component of treatment. This can be facilitated by "hot-packing" of the udder. Hot wet towels or 0.5–1 L bags of fluids

Figure 2.35: The use of a milking device to remove mastitic milk from the udder.

Figure 2.36: This image shows the placement of an intramammary tube (same mare as Fig 2.33). The teat should be wiped with alcohol and gloves should be worn.

warmed in a microwave work well. This procedure should be repeated at least every 2 hours.

- Anti-inflammatories such as flunixin meglumine should be used.
- Broad-spectrum anti-bimicrobial therapy such as trimethoprim/sulfamethazole (TMS) (30 mg/kg PO q12 h) or penicillin (20,000 IU/kg IM q12 h) and gentamicin (6.6 mg/kg IV or IM q24 h) should be used pending culture and sensitivity results.
- There is much anecdotal evidence regarding the use of bovine intramammary preparations. If used, the udder should be stripped of as much secretion as possible prior to placement and it should be borne in mind that each teat has two (and occasionally three) separate openings which lead to separate lobes. It is therefore important to place the preparation being used into both openings unless the mastitis has been confirmed to be confined to a single lobe.

Examination of the placenta (Figs 2.37–2.70)

Once passed by the mare the placenta should have a preliminary examination by foaling staff/farm manager and then be placed in a clean plastic bag or bucket with a lid and stored for further examination by the veterinarian. Preliminary examination allows recognition of potentially serious abnormalities such as gross placentitis, which would warrant an earlier than normal neonatal examination, or partial retention of the fetal membranes, which would likewise warrant early veterinary intervention on the behalf of the mare.

It is important to record all findings and the standard foaling record chart should allow for recording such information (see Appendix 2).

It is also important to remember that the placenta can carry infective organisms and as such placental examination should occur at the end of the daily routine of examining healthy prepartum mares first, followed by the foals and other mares. In addition, protective clothing such as gloves rubber boots and a plastic apron should be worn. These items should be stored on the farm and should not move from farm to farm. Staff should be educated about careful washing of hands before and after handling placentae.

Placental examination is particularly important in cases of abortion and often may provide more valuable information than fetal tissues. A special box containing all necessary equipment should be at hand should samples be required. This should contain:

- scissors
- disposable scalpel blade and handle
- sterile swabs with bacterial transport medium
- viral transport medium
- sampling bottles with 10% formalin
- plain tubes – fresh tissues are preferred in many cases and plain tubes allow for small samples to be taken which can then be held on ice during immediate transportation; plain tubes can also be used to sample amniotic or allantoic fluids.

Examination

- Weigh the placenta.
  - This should be done by farm staff as soon as the placenta is passed. It is important to weigh just the placenta and not the fetal fluids. Immediate weighing prevents errors that may occur as a result of drying or predation.
  - Normal fresh weight should be <11% of the foal's body weight.
  - A heavier placenta than normal suggests edema or inflammatory changes or an abnormal amount of fetal blood, such as may occur with early cord separation.
  - A lighter than normal placenta may be incomplete or have large avillous areas.
- Place the placenta on a wet, clean concrete surface. If placed on a dry surface it may stick. The placenta should be laid out in the shape

Figure 2.37: This image shows the correct method to lay out the chorioallantois for examination. The thicker horn of the reverse "F" is the pregnant horn, in this case the upper horn. The birth tear can be seen at lower end of the image. The somewhat unusual but not significant finding here is that the vessels are attached further along the non-pregnant horn than is normally seen. They are usually at the bifurcation of the two horns or further along the pregnant horn. Another finding here is that the two horns are of almost equal length. Short horns of equal length are generally associated with a predominantly body pregnancy, which are rarely carried to term. This placenta was from a primiparous mare that delivered a small foal (44 kg).

Figure 2.38: Normal placenta. The pregnant horn is in the lower arm of the reverse "F" in this case. It is clearly longer, thicker and more edematous. In both images a pale area can be seen in the body – this corresponds to an area of decreased villi on the reverse surface which is associated with an area of fetal contact and is not a significant finding (see Fig 2.41 and 2.42).

Figure 2.39: Another normal placenta – note the pregnant horn as the upper arm of the reverse "F" and the presence of a cystic yolk sac remnant.

Figure 2.40: Normal chorionic surface of the same placenta as in Fig 2.40. The darker areas are drying artifacts.

Figures 2.41 & 2.42: Close-up of a pale area on the allantoic and chorionic surface. These pale areas are associated with decreased villi but they are not avillous. They are normally found in the body and correspond with areas of fetal contact.

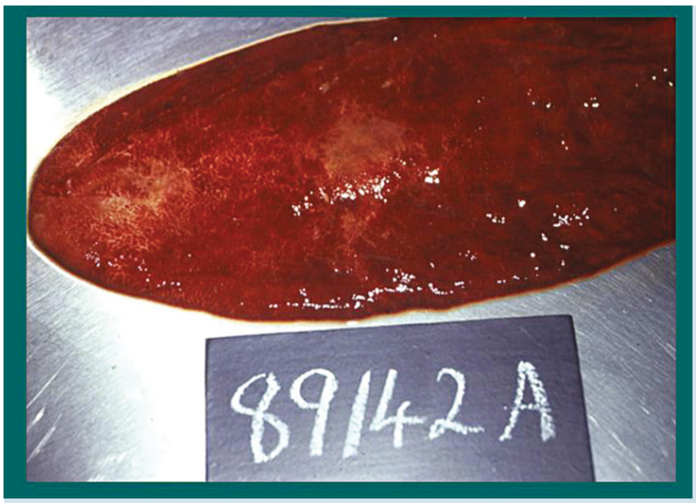
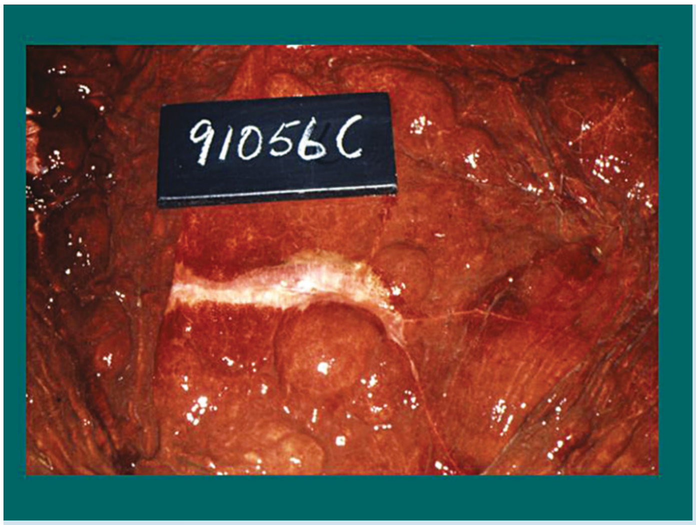


Figure 2.43: Normal chorionic surface of the tip of the pregnant horn (courtesy of the Irish Equine Centre – IEC).



Figures 2.44 & 2.45: Normal avillous areas associated with placental folds (Fig 2.45 courtesy of IEC).

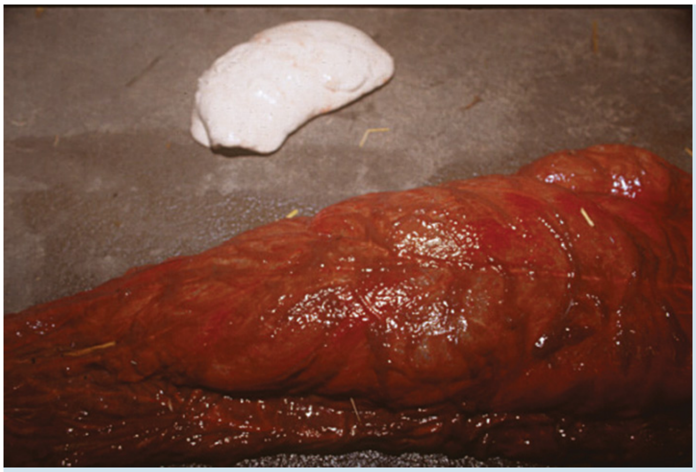


Figure 2.46: The hippomane is usually yellow/brown in color. In this case the hippomane was white but the color difference is not significant.

Figure 2.47: This image shows two hippomanes from two mares that delivered on the same farm on the same day. Again note the color difference, which is not significant.

Figure 2.48: In this image small pieces of hippomane have become attached to the allantoic surface. While this is an unusual finding it is not associated with any pathological change in this case (courtesy of IEC).

Figure 2.49: In this image a section of hippomane has again become attached to the allantoic surface but in this case has resulted in the formation of a fluid-filled pouch associated with a blood vessel and is thus pathologic (courtesy of IEC).

Figure 2.50

Figure 2.51

Figure 2.52

Figure 2.53

Figure 2.54

Figure 2.55

Figures 2.50–2.55: These images demonstrate the differing appearances of cystic yolk sac remnants. All of these are normal. Fig 2.54 is the inner surface of the cystic yolk sac imaged in Fig 2.53 (Figs 2.50 & 2.51 courtesy of IEC).

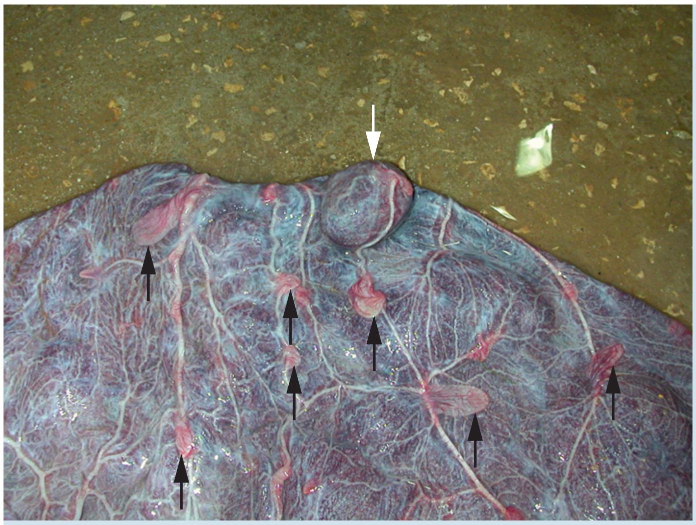


Figure 2.56: Normal endometrial outpouching (white arrow). Also note the multiple diffuse chorioallantoic vesicles (black arrows).

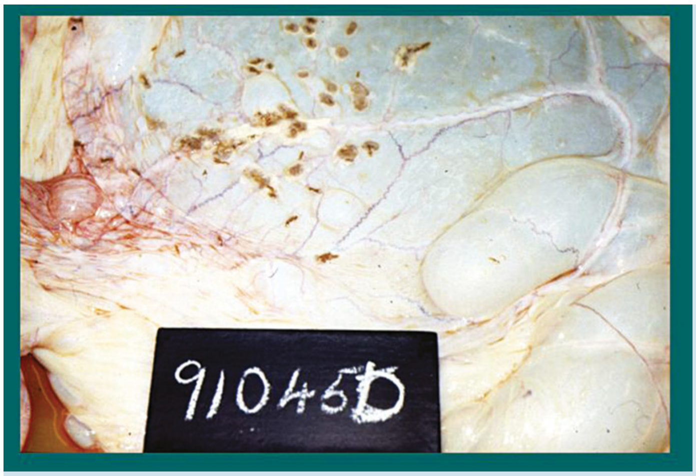


Figure 2.57: Normal mineralized deposits on the surface of the amnion (courtesy of IEC).

Abnormal findings on placental examination

Figure 2.58: Marked edema of the amnion.

Figure 2.59: Large avillous area in the body associated with *Crossiella equi* (nocardioform) placentitis.

Figure 2.60: Hemorrhagic placenta with ascending placentitis and marked necrosis in the cervical region (arrow).

Figure 2.61: Chronic placentitis. Note the thickened corrugated appearance of the placenta.

Figure 2.62: Marked bruising at the site of the birth tear. This finding indicates that the foal had difficulty breaking through the chorioallantois. Such difficulty could be associated with fetal distress or may indicate thickening of the chorioallantois.

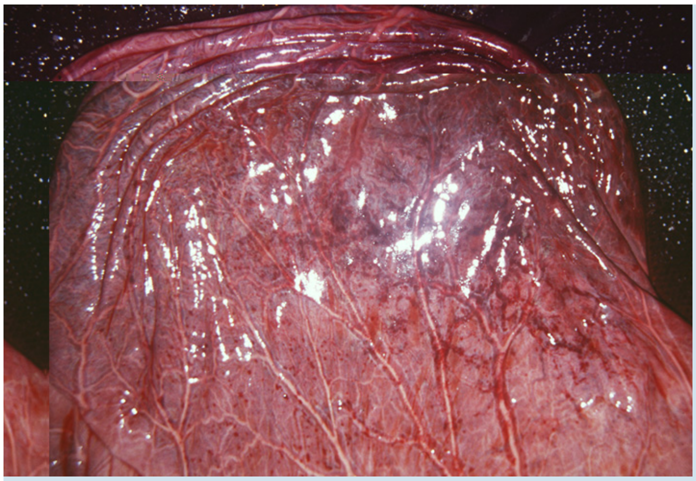


Figure 2.63: Hemorrhagic placenta.

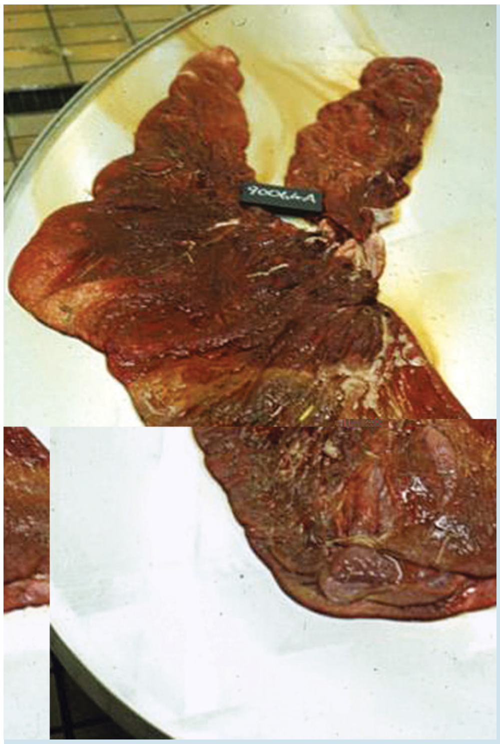


Figure 2.64: Ascending chronic placentitis. Note the thickening and discoloration of the placenta. This was also a body pregnancy as indicated by the short horns of equal length (courtesy of IEC).

Figure 2.65: Meconium staining of the amnion. Note the meconium that can be seen within the amnion.

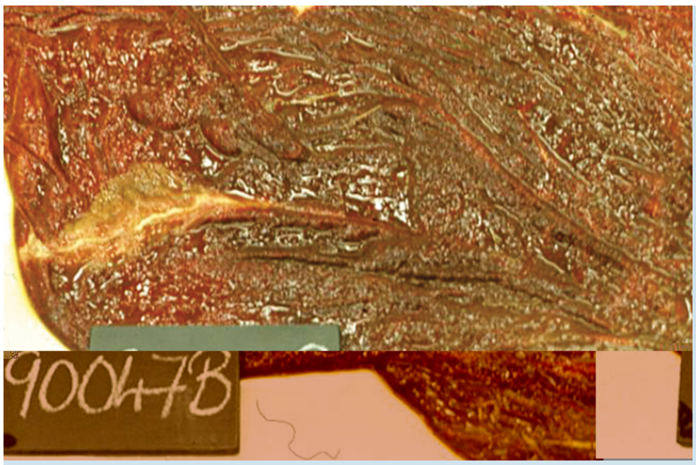
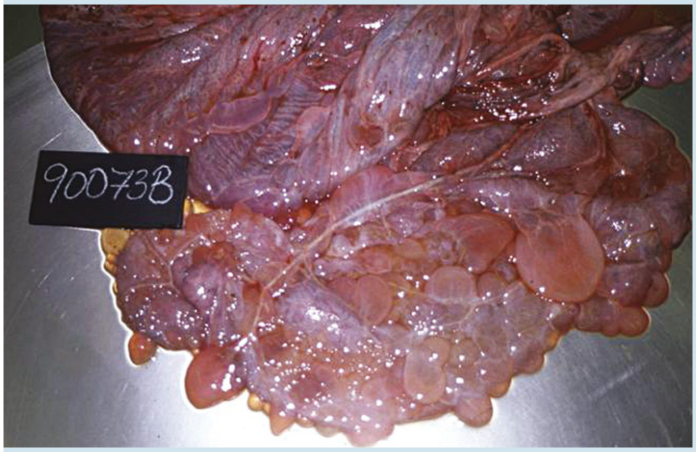


Figure 2.66: Meconium staining of the chorioallantois, note also the normal avillous area associated with a placental fold (courtesy of IEC).



Figures 2.67 & 2.68: Multiple large allantoic pouches and extensive edema. These placental changes are typically seen with fescue toxicity (courtesy of IEC).

Figure 2.69: Twin placentae of equal size. Note the large avillous area where the two placentae were in contact (courtesy of Dr. Vivienne Duggan).

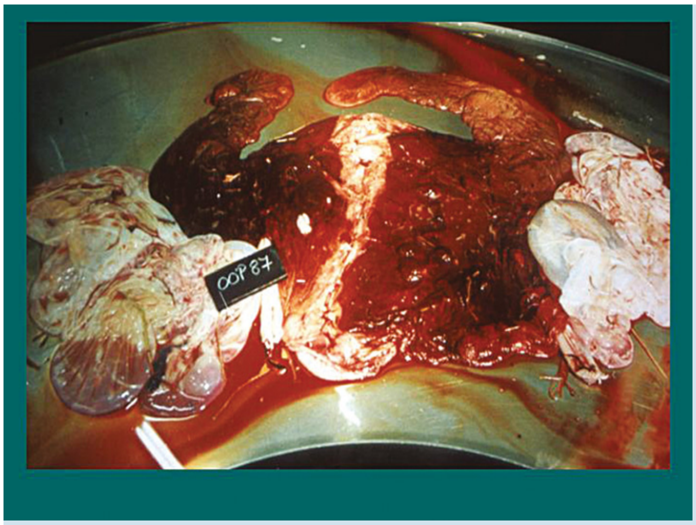


Figure 2.70: Twin placentae again of equal size showing a linear avillous area where the two placentae were in contact (courtesy of IEC).

of a reverse "F" with the thicker, wider, more edematous arm being the pregnant horn and the thinner, smaller arm being the non-pregnant horn. The lowest point of the reverse "F" is the cervical end and the cervical star and birth tear should be evident.

- The exposed (allantoic) surface should be examined first. This is the smooth gray/white surface from which the vasculature and amnion are visible. The location of the umbilical cord attachment is usually found close to the bifurcation but may be in either the pregnant or non-pregnant horn. The whole surface should be examined carefully and the amnion should also be examined before examining the chorionic surface.
- The placenta should be laid out in the same reverse "F" pattern to examine the chorionic surface which should have a uniform red-brown velvety appearance due the presence of small villi.
- Avillous areas appear as smooth pale areas and are normally evident at a number of locations:
  - at the cervical star
  - at folds of the allantochorion
  - at the sites of the endometrial cups
  - at the apex of each horn corresponding to the papillae of the fallopian tubes
  - at the sites of endometrial cysts.
- Abnormal avillous areas usually signify placental detachment. Areas of decreased villi in the body may represent areas in which the fetus was in contact.
- Normal findings of the chorioallantois are chorioallantoic vesicles and yolk sac remnants.
- The amnion has a smooth mottled gray/white appearance and is semi-translucent. Changes in color reflect meconium staining or hemorrhage. Normal findings of the amnion are mineralized deposits (usually calcium) and multiple diffuse vesicles.
- The hippomane is a soft rubbery usually brown/yellow oval shaped structure which is a composite of complex minerals (mainly from fetal urine and desquamated placental cells) and is of no clinical significance.
- The umbilical cord should be examined for bruising or other changes. The mean length of the umbilical cord in Thoroughbreds is 55 cm; longer cords are more likely to twist and shorter cords are

more likely to tear early (sometimes prepartum). Cord torsion is often wrongly blamed for fetal death and abortion. Some authors claim that greater than 12 twists is a significant finding. Varicosity or constriction of umbilical vessels and the presence of unequal twists should also be looked for. Usually there are 6–8 equal twists but in most normal situations it is not possible to count the number as the process of foaling and handling of the placenta usually results in obliteration of the normal twists. A freshly aborted fetus within its membranes provides the best opportunity for counting twists. It is also important to note that a certain amount of cord edema is normal. If in doubt sample a section of the cord for histological examination. Other normal findings of the umbilical cord are urachal dilatations and multiple diffuse tiny vesicles.

- Swabs and sections of tissue should be taken from any suspicious areas (areas with abnormalities of color, thickness or texture) and also from the area around the cervical star.

Recommended reading

Asbury AC 1993 Care of the mare after foaling. In: McKinnon A, Voss J (eds) Equine reproduction. Lea & Febiger, Philadelphia, p 976–980.

Frazer GS 2003 Post partum complications in the mare. Part 2: Fetal membrane retention and conditions of the gastrointestinal tract, bladder and vagina. Equine Veterinary Education 15(2):91–100

LeBlanc MM 1999 Disease of the uterus. In: Equine medicine and surgery. Mosby, St Louis, p 1165–1173.

LeBlanc MM. Disease of the vagina, vestibule and vulva. In: Equine medicine and surgery. Mosby, St Louis, p 1175–1193

Steel CM, Gibson KT 2001 Colic in the pregnant and periparturient mare. Equine Veterinary Education 13(2):94–104

Vaala W et al 2002 Initial management and physical examination of the neonate. Examination of postpartum mare and placenta. In: Smith B (ed) Large animal internal medicine. Mosby, St. Louis, p 291–292

CHAPTER 3

Neonatal examination, clinical procedures and nursing care

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Introduction

Delivery of a live foal not only provides a sense of relief but also may provide a misguided sense that all is now well – especially when the foal stands and nurses. Many neonatal foals may appear to be completely normal and within a few hours can be severely compromised. Initial clinical signs of many disorders may be vague and go unnoticed at farm level. All owners should therefore be advised to have their neonatal foals examined within 12–24 hours of birth.

The approach to foal management will vary depending on the size of the breeding establishment involved (individual mare owner versus stud farm), the economic value of the foal, and environmental, geographical and seasonal factors. All of these factors should be taken into account when advising each owner what is the best type of examination procedure for their foals. For example owners of valuable foals may prefer additional diagnostics to be performed at the time of initial examination.

The use of "risk categories" for foals is also useful for both owner and veterinarian in determining the components of the examination and the stage and speed of intervention. A farm which has a history of high neonatal illness or a current disease outbreak would warrant earlier than normal neonatal examinations, specific diagnostics and rapid interventions.

The use of examination forms (Appendix 1) is useful as it may guide future decision making and can also be useful if the animal is later assessed by another practitioner. Owners should also be encouraged to keep foaling forms (Appendix 2) which may provide vital information for the examining clinician. Frequently a vital piece of history may not be relayed orally.

Risk categories

Low risk (Figs 3.1–3.4)

A foal can be regarded as "low risk" if it meets the following criteria:

- No maternal, neonatal or environmental risk factors have been identified (see below).
- Gestation was of normal duration:
  - pony breeds 320–345 days
  - Thoroughbred 320–360 days
  - donkeys 360–380 days.
- Normal parturition; stage 2 labor lasted no longer than 20 minutes and no significant manipulation of the foal was required for delivery.
- Normal postpartum events:
  - foal stood within 2 hours of delivery
  - foal nursed within 3 hours of delivery.
- Placenta is visually normal and <11% of weight of foal.

Low risk foals should receive a thorough clinical examination and determination of serum IgG status. Any abnormalities found during the examination should guide the selection for further diagnostics.

Moderate and high risk foals

Moderate risk foals have been described as foals that have only one risk factor of maternal, environmental or foal origin. These foals if not

Figures 3.1–3.4: Normal parturition. Fig 3.1 shows two feet and a nose at the vulva – one leg is always more advanced than the other to allow easy passage of the shoulders which are the widest part of the foal. Fig 3.2 shows the mare now in a recumbent position and the foal has now been passed to mid-cervical level. Fig 3.3 shows the foal with just the lower half of the hind legs remaining within the vagina. The umbilical cord is still attached but the amnion has been removed from the foal. Fig 3.4 shows the mare nickering to her foal. The mare and foal should not be disturbed at this point.

treated correctly can quickly become high risk and as such both categories should initially be approached in the same manner. High risk foals are those that have more than one risk factor.

Many of the risk factors involved are identified before delivery and allow for anticipation of problems and warrant early intervention, e.g. maternal illness or concurrent infectious disease on the premises. In addition to the clinical examination outlined below, which will take place at an earlier stage following birth, additional measures should be taken:

- Determine if chemoprophylaxis is warranted:
  - leukopenia (<5,000 cells/μL)
  - placental pathology
  - foal with failure of passive transfer
- Frequent monitoring of the foal for signs of deterioration or absence of normal developmental steps, e.g. failure to nurse, urinate etc
- Serum chemistry with electrolyte evaluation
- Serial monitoring of CBC
- Serum IgG levels – IgG may be rapidly consumed in these foals and repeat analysis is advisable
- Frequent monitoring of body weight – foals should gain 1–1.5 kg daily.

Maternal risk factors (Figs 3.5–3.9)

- Dystocia
- Caesarian section

- Partial or complete premature placental separation
- Medically induced labor
- Parturition prior to 320 days' gestation
- Placentitis
- Concurrent illness or fever
- Recent medical or surgical event
- Recent transport stress
- Twin pregnancy
- Vaginal discharge
- Chronic lameness or incoordination
- Premature lactation
- Agalactia
- Prolonged gestation with oversize foal (uncommon)
- Pelvic abnormalities
- Drug related; long term use of non-steroidal anti-inflammatories.
- Previous history of dystocia, or delivery of septicemic or neonatal isoerythrolysis foal.

Neonatal risk factors (Fig 3.10)

- Twins
- Premature / dysmature
- Death of the dam
- Meconium staining
- Foals which do not rise and nurse by 3 hours of age
- Failure of passive transfer.

Environmental risk factors

- Foaling in contaminated area
- Foaling in cold or wet conditions
- Infectious disease on the premises
- Disrupted foaling.

Figure 3.5: Redbag – premature placental separation which can be expected to result in decreased oxygen supply to the foal during delivery.

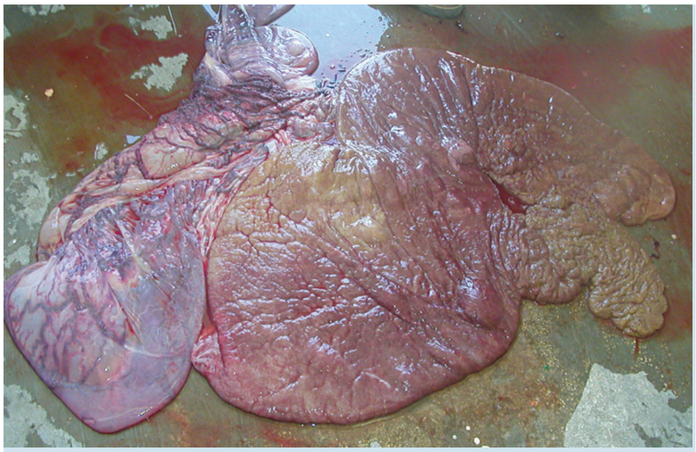


Figure 3.6: Placentitis – a thickened discolored placenta with poor development of the horns indicating a body pregnancy. Body pregnancies are rarely carried to term. The foal in this case was born alive 1 month pre-term.

Figure 3.7: Twins. These twins were delivered alive and healthy. Both twins rarely survive and if so they are both usually smaller than other foals of their age group and breed.

Figure 3.8: Previous pelvic fractures have resulted in a grossly deformed pelvis in this mare. Pelvic fractures frequently result in narrowing of the pelvic canal. This mare had an elective C-section.

Figure 3.9: Premature lactation. This mare ran milk for 6 weeks prior to foaling. Ultrasound examination of the uteroplacental unit indicated thickening and separation of the placenta.

Figure 3.11: IUGR foal demonstrating typical disproportionate head-to-body size. Also notice the floppy ears which are typical of premature foals.

Figure 3.10: Meconium staining of a newborn foal's hooves.

Figure 3.12: Tendon and joint laxity in a premature foal.

Routine examinations

All neonatal examinations should commence with a thorough examination of the placenta (see Chapter 2, p. 35). Initial assessments of maturity and behavior should be made at a distance with any abnormalities warranting closer examination before proceeding with the remainder of the clinical examination.

Maturity (Figs 3.11–3.19)

- Gestational length of normal duration is not a good indication of individual readiness for birth. Dysmature foals are those with a normal gestational age but they have signs and behavior consistent with prematurity. Since premature and dysmature foals show the same clinical signs they can both be discussed under the heading "unreadiness for birth."
- In-utero growth retardation (IUGR) results in the birth of small foals which may or may not show signs of prematurity. Common causes of IUGR are placentitis, in utero infections and twinning.
- Signs of "unreadiness for birth":
  - *Small size for breed or gestational age*, with lower than normal birth weight. Such foals often appear to be "thin." Many are also born dehydrated, which can be a reflection of placental insufficiency, metabolic derangements or in-utero infections.

Figure 3.13: This foal was born at 303 days of gestation and although it looked relatively mature for its gestational age (long hair coat, relatively erect ears) it had laxity of its flexor tendons and generalized weakness. It needed assistance to stand and was unable to nurse. The mare had been treated for placentitis for 22 days prior to delivery.

Figure 3.14: Dorsopalmar radiographs of the carpal joints demonstrating Grade 1 incomplete ossification. Note the absence of many of the cuboidal bones in addition to the proximal epiphysis of MC3.

- "*Weakness.*" Slow to stand, poor sucking reflex. Poor muscle development in addition to laxity of the flexor tendons are commonly seen. Poor maternal recognition and environmental awareness are also common.
- *Incomplete ossification of the tarsal/carpal bones* results in further difficulty standing. Weight bearing in such foals can result in crushing of the tarsal/carpal bones leading to permanent deformities. All foals that are either known to be premature or have the appearance of dysmaturity should have carpal and tarsal radiographs performed as early as possible. Poor bone development may preclude an athletic career and thus may alter the course of treatment.

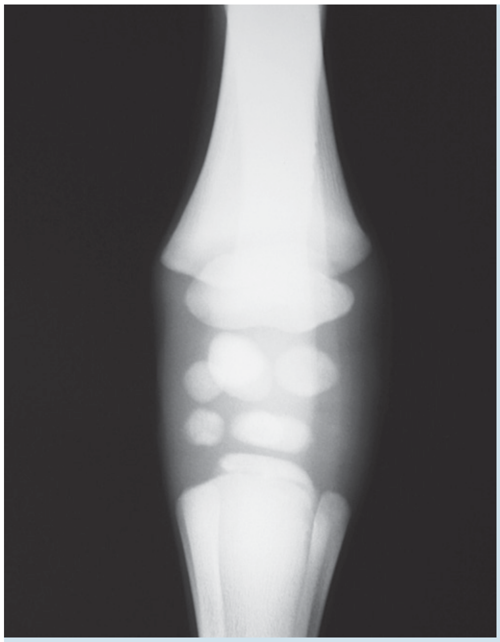


Figure 3.15: Dorsopalmar view of the carpus from the foal in Fig 3.13 again demonstrating incomplete ossification of the carpal bones. This is a Grade 2 (SOI); there is evidence of ossification of all carpal bones and the proximal epiphysis of MC3 is present but still open.

Figure 3.16: Fine silky haircoat of a premature foal.

- *Fine, silky haircoat with soft ears and lips.*
- *Domed forehead* is a common sign but is not exclusive to these foals and can also be seen with hydrocephalus and certain breeds.
- Mucous membranes may be paler than normal but the *tongue can have a marked red/orange appearance.*
- *Synechium present.* "Slipper hooves," where hooves do not dry out and separate normally.
- *Increased susceptibility to hypothermia* with many foals having a lower than normal body temperature. Glycogen stores in these foals have often failed to develop or are rapidly depleted.

Figure 3.17: Synechium – "slipper hooves."

Figure 3.18: Premature foal. Note the entropion and brightly colored tongue.

Figure 3.19: Domed forehead in a premature foal.

- *Reduced tolerance to oral feedings*, with postprandial colic being a common feature. Intestinal motility may be absent (ileus) or may be excessive and incoordinated. Smaller than normal enteral feeds are often required for these foals and parenteral nutrition is required for many that do not tolerate enteral feeding.
- *Abnormal respiratory pattern*. This may reflect fetal atelectasis (failure of lung expansion with decreased surfactant), which is characterized by an increased abdominal component or central respiratory depression.

Laboratory findings in foals showing signs of "unreadiness" for birth

- Adrenocortical insufficiency:
  - decreased (<1) or reversed neutrophil : lymphocyte ratio – normal is 2 : 1 after 2 hours
  - leukopenia with lymphocytosis (>4.0×10^9/L) and neutropenia (<1.0×10^9/L)
  - low plasma cortisol (not useful in practice due to the time delay in analyzing samples)
  - failure to respond to ACTH stimulation test
- Macrocytic, normochromic anemia may be seen
- Decreased blood glucose (<60 mg/dL or 2.5 mmol/L) at 2 hours post-foaling
- Low serum IgG which may be the result of failure to ingest colostrum or poor intestinal absorption; such foals usually have increased susceptibility to infection
- Blood gas abnormalities:
  - venous acidosis, pH <7.25 (normal pH >7.3)
  - evidence of hypoventilation – increased $PaCO_2$ and decreased PaO_2.

Behavior (Figs 3.20–3.22)

This should be observed from a distance initially and should include the following:

- Does the foal recognize and have affinity for its dam? Most normal neonates if not already standing when disturbed will rise to their feet and immediately go to the mare's side.
- Does the foal appear to be able to nurse normally? (Assessment of the adequacy of the colostrum / milk supply may be difficult in the early stages with a healthy foal as it will nurse the mare frequently and her udder should appear empty.) A foal that makes frequent attempts to nurse and appears to be frustrated when doing so may be an indication of a problem. A full distended udder is an indication that the foal is not nursing sufficiently.
- Does the foal appear to be able to see normally? Not walking into walls, people etc.

Figure 3.20: Normal behavior with foal showing affinity for its dam.

Figure 3.21: Normal suckle reflex: note the curling and protrusion of the tongue.

Congenital abnormality

Observe for any apparent congenital abnormality or injury; e.g. milk at the nares may be an indication of a cleft palate.

Size and maturity

Evaluate the size and maturity of the foal taking account of stated gestational age. All foals should be weighed as changes in body weight are good indicators of clinical improvement or deterioration. It is obviously impossible to assess weight gain over the first few weeks if the starting weight is not known. Foal weight should ideally be determined after the clinical examination is complete; e.g. if fractured ribs are discovered during the examination then the foal should be not be moved from the box and excessive manipulation should be avoided.

Figure 3.22: Foal in Fig 3.13 demonstrating an absence of a suck reflex.

Figure 3.23: Foal restraint by holding both ears. It is important not to tug or aggressively pull on the ears as this may cause transient nerve damage.

Clinical examination (Figs 3.23–3.26)

Following observation from a distance the foal should be correctly restrained for the remainder of the examination.

Vital signs

Respiratory rate

- Respiratory rate and pattern should be assessed from a distance as restraining / handling may significantly increase the respiratory rate. A respiratory rate of >60 BPM is normal immediately after birth but falls to 20–40 BPM within 1–2 hours of birth.
- Any irregularity of pattern and a high or low respiratory rate is significant. Abnormal chest or abdominal movements associated with respiration may be related to such conditions as rib fractures, other thoracic trauma or diaphragmatic abnormalities. High respiratory rates can be related to several systemic conditions in addition to excitement so careful clinical examination is critical.
- Any nasal discharge is generally significant in the newborn foal. Meconium may be seen at the nares following meconium aspiration. Milk may be seen at the nostrils and can be related to congenital defects such as cleft palate or pharyngeal paralysis (part of the Hypoxic Ischemic Encephalopathy (HIE) syndrome).
- Following restraint the chest should be auscultated thoroughly. Bronchovesicular sounds are louder than those of adults because of the decreased muscle mass over the thorax compared to the adult horse.
- Any abnormality detected at any stage of the examination warrants further investigation (arterial blood gas evaluation, ultrasound examination, radiography).
- The patency of the airway should be assessed first in any respiratory distress situation. Placing a hand at each nare allows assessment of airflow.

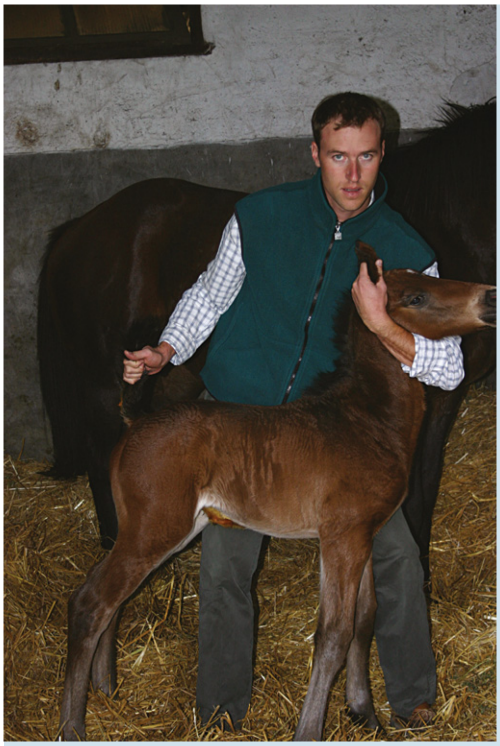


Figure 3.24: Foal restraint by holding one ear and base of the tail. It is important to hold the ear that is furthest from the handler with the head stabilized against the handler's body. Ideally the tail should be grasped at the base (lower than this image).

Temperature

- Hypothermia can be associated with severe systemic disease, cold environment or poor technique in taking the rectal temperature.
- If a hypothermic reading is recorded, re-check the result and palpate the extremities, which should feel cold if the foal is indeed hypothermic.
- Hypothermia associated with severe systemic disease is usually accompanied by many other signs and such foals are frequently moribund.
- If hypothermia is deemed to be due to an extremely cold environment, the foal should be warmed and serum glucose levels should

Figure 3.25: How to pick up a foal. Foals should not be picked up by placing pressure on the sternum. Grasping the foal around the chest and buttocks results in a relaxed state.

Figure 3.26: How to place a foal in lateral recumbency. The tail is grasped in one hand and the head is held above the muzzle with the other hand. The foal is then gently folded in on itself. There is usually no resistance to this procedure. This is the best method to use as the foal leans against the handler's body and is gently placed on the ground. A foal should not be knocked down by pulling its legs out from under it as it is likely to hit the ground hard.

be assessed without delay as hypoglycemia frequently occurs concurrently with hypothermia as the foal uses its limited available reserves in an attempt to maintain body temperature. Additional warmth may be provided by means of special foal blankets/rugs, heat lamps and heating pads.

- An elevated rectal temperature is significant and is an indication of infection and toxemia. Severe pain (associated with limb or rib fractures) may also cause an elevation in rectal temperature.

Pulse

- The normal heart rate immediately after delivery is 40–80 BPM. The heart rate will rise to 130–150 BPM within minutes of birth and then gradually falls over the first week of life to 60–80 BPM. Excitement and handling will cause elevations in heart rate.
- The pulse can be palpated in the facial, metatarsal and median arteries with the fingertips. It should be readily detectable.

Cardiovascular system (Figs 3.27–3.32)

Examination of the cardiovascular system should include the following.

Heart auscultation

Auscultation of the foal heart is made easy by the proximity of the heart to the chest wall and the lack of body fat. The sensitivity of auscultation for identification of congenital heart disease, significant valvular disease and persistent cardiac arrhythmias is high. The sensitivity of auscultation for identification of myocardial or pericardial diseases is lower unless other abnormalities are also present.

Cardiac murmurs are classified according to a grading system (Table 3.1), their location in the cardiac cycle (systolic, diastolic or continuous), timing in the cardiac cycle (early, mid, late or holo-), shape or frequency, their point of maximal intensity (PMI) and radiation. Systolic murmurs occur anytime between the first and second heart sounds. Diastolic murmurs occur between the second and first heart sounds. Continuous murmurs occur throughout the cardiac cycle. The PMI usually corresponds to the location of one of the heart valves.

- Murmurs associated with the tricuspid valve are commonly located in the third or fourth intercostal space on the right-hand side.

Table 3.1: Grading of cardiac murmurs

Grade 1	Soft murmur heard after a number of minutes of careful auscultation
Grade 2	Soft murmur that is heard immediately on auscultation
Grade 3	Murmur of moderate intensity
Grade 4	Loud murmur associated with a slight palpable thrill
Grade 5	Loud murmur which is not heard when the stethoscope is removed from the chest
Grade 6	Loud murmur which is still audible when the stethoscope is moved away from the chest

Figure 3.27: Heart auscultation.

Figure 3.28: Normal pink mucous membranes.

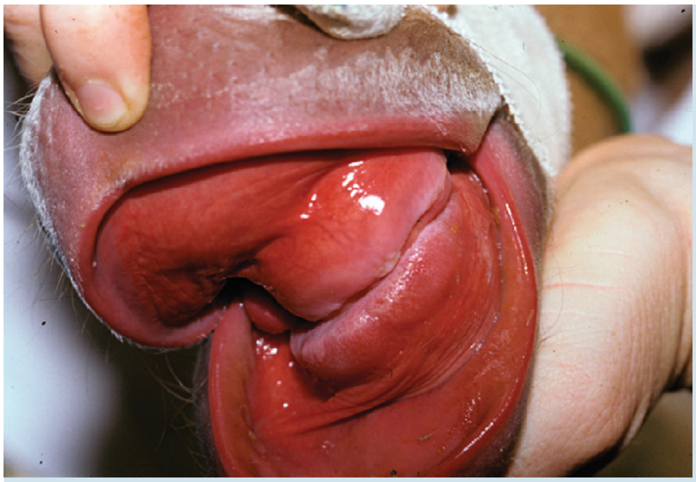


Figure 3.29: Hyperemic mucous membranes typical of septicemia.

Figure 3.30: Episcleral hemorrhage frequently seen in foals with a history of dystocia.

Figure 3.31: Petechial hemorrhages on the ear pinna in a septicemic foal.

Figure 3.32: Severe icterus in a 1-day-old foal diagnosed with neonatal isoerythrolysis.

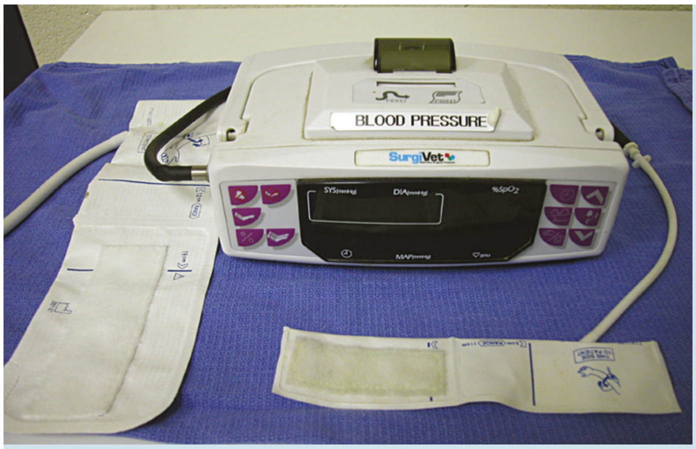


Figure 3.33: Indirect blood pressure monitor – note the different size tail cuffs.

Figure 3.34: Abdominal auscultation.

- Murmurs associated with the mitral valve will be heard best in the fifth intercostal space on the left-hand side just dorsal to the level of the elbow. These murmurs frequently radiate dorsally.
- Murmurs associated with the aortic valve are commonly heard in the fourth intercostal space on the left-hand side just caudal and dorsal to murmurs associated with the pulmonic valve which are best heard in the third intercostal space on the left-hand side.

A holosystolic grade I–IV murmur over the left heart base is a common finding in foals up to 1 week of age and is normal (until closure of the ductus arteriosus).

Loud murmurs with palpable thrills in neonates are associated with cardiac defects (see Chapter 7).

Mucous membranes

- Mucous membranes should be pale pink and moist with a capillary refill time of less than 2 seconds. They are however only a subjective indicator of the degree of oxygenation of the blood.
- It is normal to have mild episcleral hemorrhages associated with pressure during passage through the birth canal. These can be marked following dystocia (see Fig 3.30).
- Petechial hemorrhages are abnormal if found on the gums, tongue, palate, vulva or ear pinnae.
- Severe icterus in the first few days is most commonly related to neonatal isoerythrolysis. Icterus associated with liver disorders is generally seen in older foals.
- Hyperemia is an indicator of sepsis and warrants further investigation, e.g. CBC, fibrinogen and serum biochemistry.

Jugular pulses

- Jugular pulses are abnormal. The jugular veins should fill quickly if the foal is not hypovolemic.

Blood pressure (BP) (Fig 3.33)

- Assessment of blood pressure is not usually performed in the apparently healthy foal.
- Non-invasive methods using tail or limb cuffs are available. Day 1 systolic BP in lateral recumbency should be 81 ± 10 mmHg. Day 7 systolic BP in lateral recumbency should be 104 ± 21 mmHg.

Gastrointestinal system (Fig 3.34)

- Examine the oral cavity for cleft palate and bite defects.
- Observe nursing behavior and swallowing. Abnormal swallowing with milk at the nostrils is a good indicator of pharyngeal paralysis.
- Meconium staining is an indicator of in utero stress. This may or may not be observed depending on the time at which the foal is examined. If present, meconium may stain the skin / coat or may be observed in the medial canthus of the eye, the nares or the mouth.
- The caretaker should be questioned as to the passage of meconium, its quantity and color.
- Abdominal auscultation:
  - borborygmi are normally present in all four abdominal quadrants
  - 'splashing sounds' may indicate impending diarrhea.
- The anus should be examined to rule out atresia ani.
- Foals are notably intolerant of abdominal pain and will demonstrate signs of severe colic with relatively mild abdominal distension.

Differentials for abdominal distension (usually with severe pain) in neonates are:

- meconium impaction
- atresia ani or coli
- impending enteritis/colitis
- volvulus of small intestine or large intestinal torsion
- intussusception
- ileocolic aganglionosis (lethal white syndrome).

Differentials for abdominal distension (usually with mild to moderate pain) in neonates are:

- uroperitoneum
- peritonitis
- gastric distension associated with over feeding and/or ileus in premature or seriously ill foals.

Urogenital system (Fig 3.35)

- External genitalia should be examined for congenital abnormalities.
- Scrotal hernias are not uncommon in the first 24–48 hours but should be of small size. It is important to differentiate this from fluid accumulation within the scrotum e.g. uroperitoneum or peritonitis.

Figure 3.35: Umbilical clamp in place to prevent excessive hemorrhage which may be caused by manual tearing or cutting of the umbilical cord.

- Urination should occur by 8 hours and should be observed as a full flow of urine rather than dribbling.
- The umbilicus should be carefully examined with a gloved hand:
  - Any enlargement, heat or swelling should be noted and warrants further examination (ultrasonographic examination).
  - The urachus may remain patent for 12–24 hours. These foals should be carefully assessed for sepsis.
  - A patent urachus may appear as a few wet hairs in the umbilical region or can appear as a full stream of urine during urination. Cord clamping or cutting increases the incidence of urachal patency.
- Manual tearing of the cord can cause umbilical hemorrhage which may result in an intra- or extra-abdominal bleed. The resultant blood loss can be severe.
- Examine carefully for the presence of an umbilical hernia.
- Routine umbilical care should be discussed with the client (see p. 76).

Ophthalmic system

- Note any congenital abnormalities, e.g. micro-ophthalmia. Examination of the eyelids to detect entropion should be performed prior to manipulation of the periorbital region.
- Examine the eyes for evidence of ulceration or uveitis. ***Corneal sensitivity is poor in foals and they can have extensive ulceration with little outward signs of discomfort.***
- Pupils should be large, equal and circular in 1-day-old foals.
- Pupillary light reflexes (PLRs) are normally present at birth but are unreliable as they can be sluggish in excited foals and do not necessarily correspond with intact vision (see Fig 3.46).
- The menace response is lacking in normal foals until at least 5–10 days and for as long as 2 weeks in some foals (see Fig 3.47).

Musculoskeletal

Rib fractures (Figs 3.36 & 3.37)

These are relatively common in newborn foals and the ribs should be carefully palpated, especially at the costochondral junction. A single rib or many ribs (resulting in “flail” chest) may be affected. The incidence increases in cases of dystocia and foals with a known history of dystocia should be examined radiographically or ultrasonographically as non-displaced fractures may be difficult to palpate.

Figure 3.36: Palpation of the rib cage to check for rib fractures.

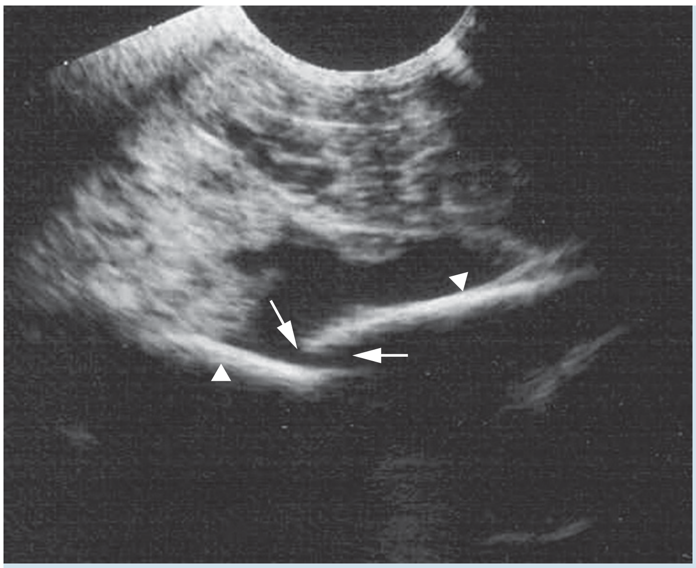


Figure 3.37: Ultrasonographic image of a rib fracture demonstrating the fracture (arrows) and fluid accumulation at the fracture site. The rib is the hyperechoic line (arrowheads).

Figure 3.38: Severe joint contracture in a newborn foal which necessitated euthanasia. Severe joint contrachure commonly results in dystocia, as in this case.

Figure 3.39: Severe contracture of the flexor tendons in a newborn foal.

Contracted tendons (Figs 3.38 & 3.39)

The approach depends on the degree of contracture. Mild cases (heel just off ground) may improve with minimal intervention over the first few days of life. More severe cases (foal standing on toe or dorsal surface of hoof wall) require intervention in the form of splinting, casts and administration of oxytetracycline.

It is important to differentiate tendon contracture from joint contracture which has a much poorer prognosis. Such limbs cannot be manipulated into a normal position and overzealous efforts to do so may result in limb fracture.

Weakness or laxity of flexor tendons (Fig 3.40)

More common in premature/dysmature foals but can occur in full-term otherwise normal foals. If mild (greater than normal fetlock drop but toe is still on the ground), improvement can be marked with a regime of limited exercise. More severe cases (toe pointing upwards with foal walking on the bulbs of its heels), may require intervention. Commercial or homemade heel extensions are very helpful and can radically improve ambulation. *A common misconception is that such foals should be bandaged to protect their legs – this will only make the condition worse.*

Figure 3.40: Laxity of the flexor tendons. Flexor laxity will always be seen in premature foals but may also occur in term foals.

Figure 3.41: Scoliosis in a newborn foal.

Abnormalities of the axial skeleton (Figs 3.41 & 3.42)

Abnormalities (scoliosis, kyphosis, curvature of the spine) may occur alone or be associated with other deformities, e.g. contracted tendons.

- Over-critical assessment of conformational abnormalities should be avoided in the first few days as many foals show significant improvement in the first week of life.

Figure 3.42: Kyphosis and scoliosis in a young foal.

Figure 3.43: Normal submissive "chewing" movements.

Integumentary system

The skin of the normal foal is soft and pliable. The hair coat should be of uniform length and consistency. Premature/dysmature foals have abnormally thin skin and short silky haircoats. A thin haircoat may also be associated with hypothyroidism. Congenital skin defects can occur but are rare (see Chapter 12).

Neurological examination (Figs 3.43–3.47)

Normal newborn foals are inherently incoordinated with apparently ataxic movements – in part because of the relative immaturity of the cerebellum and upper motor neuron system. This makes neurological examination of the newborn somewhat difficult and significantly different to adults. Routine assessment of neurologic function should include the following.

Figure 3.44: Assessment of withdrawal reflex in a newborn foal. Note the extension of the opposite limb.

Affinity to and recognition of the dam

Loss or absence of this may be an early indicator of neurological dysfunction but may indicate other problems such as depression associated with sepsis or maternal aggression and associated fear on behalf of the foal.

Is the foal blind? Determining if a foal is actually blind can be challenging. Most blind foals will stay close to their dams and will be able to locate their dam through smell or vocalization. Holding the foal while walking the dam a short distance away and then releasing the foal to determine if it can locate the dam can be useful. However, care should be taken as foals may become panicked and if they do not locate the dam may collide with other objects or even fall over. Such an examination should only be performed in a confined safe place.

Behavior

Normal healthy foals are bright, alert and responsive to external stimuli. Premature/dysmature foals may be less bright and responsive than normal/full term foals. The newborn foal is usually standing within 2 hours of birth, is cognisant of its surroundings and locates its dam and nurses.

Effective feeding is a complex process which involves a proper swallow (cranial nerves IX, X, XI) reflex, movement of the lips (VII), jaw (V) and tongue (XII) as well as recognition of the dam's udder (II, cerebrum).

Newborn foals have a more flexed head posture than adults and "bobbing" head movements may be normal for the first few days. Continuation of exaggerated head movements with an intention tremor is highly suggestive of cerebellar abnormality.

Normal foals will resist handling and restraint. However, restraint by grasping the foal around the chest and buttocks will result in a relaxed state (see Fig 3.25).

Convulsions or seizures in newborn foals may take a variety of forms and can be subtle. Lip smacking, blinking and random nystagmus may be seen. Overt seizures are usually manifested by bruxism, odontoprisis, chewing/salivating, paddling of limbs and opisthotonus (see Figs 15.21–15.26). These should not be confused with the normal chewing action seen as submissive behavior in many foals (Fig 3.43).

Figure 3.45: Assessment of the patellar reflex in a newborn foal.

Figure 3.46: Assessment of PLR in a 2-day-old foal. PLRs are normally present at birth but are unreliable as they can be sluggish and do not necessarily correspond with intact vision.

Figure 3.47: Lack of menace response in a 3-day-old foal – this response is not normally evident until at least 5–10 days of age.

Posture and gait

Similar to other newborns, foals have increased range of limb movement as compared to adults. "Abnormal" postures may indeed be "normal". The neonate's stride is short, rapid and dysmetric with exaggerated steps which slap on the ground. Nervous control of the limb muscles develops quickly (usually in 24–48 hours) and results in postures more closely resembling those of an adult. Foals which are confined to their stall or those which spend a long time recumbent may be slower to develop the adult type stance and gait.

Cranial nerve examination

All cranial nerves should be fully functional within hours of birth and assessment is similar to adults with the exception that foals have jerky withdrawal reflexes.

Trigeminal (V) and facial nerve (VII)

The ear, eyelid, lip and nostril reflexes are present at birth (V sensory and VII motor). Stimulation will result in withdrawal.

Vestibulocochlear (VIII)

Foals can hear at birth. Normal physiological nystagmus that occurs when the head is moved from side to side is present at birth.

The presence of nystagmus with the head in a normal position (spontaneous) or with the head held in various abnormal positions (positional) indicates dysfunction of the vestibulocochlear nerve. Other common signs of VIII dysfunction include head tilt and circling.

With peripheral VIII dysfunction (involving the inner ear) the nystagmus is consistent regardless of head position and the direction (slow phase) is towards the side of the lesion and towards the direction of the head tilt. The nystagmus is usually horizontal but it can be rotary or arc shaped.

With central VIII dysfunction (associated with disorders of the caudal brainstem or medulla) the nystagmus is inconsistent in direction and may vary as the head position changes. Vertical nystagmus may be seen with central or bilateral peripheral lesions (rare). Signs of depression and involvement of other cranial nerves also indicate central dysfunction.

Glossopharyngeal (IX), vagus (X) and accessory (XI)

These nerves supply the pharyngeal and laryngeal muscles that control swallowing. They can be assessed by observing normal swallowing or by passage of a nasogastric tube. If in doubt, endoscopy can be used to assess pharyngeal and laryngeal movement. Dysfunction in newborn foals is commonly seen as part of the HIE syndrome or may be associated with other central disorders. Dysfunction in older foals can also be related to disorders of the guttural pouches.

Hypoglossal (XII)

Supplies the muscles of the tongue. Many normal foals will hang their tongue out of the mouth. This can be regarded as normal as long as it is withdrawn when stimulated. Central lesions may also result in tongue protrusion, as can severe systemic disease.

Limb reflexes

In foals less than 3–4 weeks of age, flexor or withdrawal reflexes cause prominent extension of the contralateral limb. Patellar and triceps reflexes are exaggerated in foals up to 10–14 days of age. Sick foals may show a delayed waning of the exaggerated reflexes.

Tests and interventions

Assessment of colostral quality (Figs 3.48–3.50)

Figure 3.48: Good quality colostrum (thick and dark yellow) on the right vs poor quality colostrum (resembles milk) on the left.

Figures 3.49 & 3.50: Colostrometer on left for assessment of colostrum specific gravity. Brix refractometer on right for assessment of total sugars in colostrum.

- Colostrum can be subjectively assessed on the basis of appearance. A thick, yellow sticky secretion is most likely of good quality, whereas a dilute, white or translucent secretion is most likely inadequate. Subjective assessments should not be used alone.
- More objective assessments are estimations of specific gravity (>1.065), sugar refractometry (>20%) or determination of IgG concentration (>70 g/L).
- Specific gravity can be determined with a colostrometer.
- Handheld refractometers designed for measurement of sugar concentration of solutions are easy to use. Table 3.2 shows the relationship between sugar percentage and IgG concentration.
- Determination of IgG concentration can be performed at a laboratory using the single radial immunodiffusion test (SRID) or by the use of a variety of commercially available kits, which are usually based on glutaraldehyde precipitation or latex agglutination. Such kits are

Table 3.2: Colostral quality determined by refractometry

BRIX (%)	IgG CONC. (g/L)	COLOSTRAL QUALITY
10–15	0–28	Poor
15–20	28–50	Borderline
20–30	50–80	Adequate
>30	>80	Very good

easy to use and tests can be performed stable-side. The SRID test takes 24 hours to complete and thus is most suitable for colostrum that is to be banked.

Colostrum bank

- Forming a store of colostrum is advisable in order to supplement future foals whose mothers have either inadequate colostrum quality or quantity. Central facilities such as veterinary clinics will often have colostrum banks.
- Colostrum for a colostrum bank should always have an IgG concentration >70 g/L. Assessment of the IgG concentration should ideally be performed by SRID.
- Storage in a "domestic" freezer will retain the IgG concentration for 12 months. The concentration of immune proteins (complement) will decrease over time.
- Storage at −70°C will result in permanent maintenance of all components.
- Normal low-risk foals will require 0.5–2 L of colostrum depending on quality, which may be made up of colostrum from several mares.
- Colostrum of a mare which has produced a neonatal isoerythrolysis (NI) foal should never be placed in a colostrum bank. Colostrum should always be tested for IgG and common antibodies against red blood cells.

Poor or inadequate colostrum

The ingestion of poor quality colostrum or failure to ingest an adequate volume of colostrum can result in failure of passive transfer (FPT) which is a well recognized risk factor for sepsis (see Chapter 11, p. 297).

Determination of serum IgG status

Sampling the foal's blood should be performed when the routine foal examination is being carried out at 12–24 hours. By 18–20 hours the ability of the intestine to absorb ingested colostrum is virtually zero so corrective measures after this time involve the administration of intravenous plasma. Thus it is advisable where possible to perform the neonatal examination and blood sampling closer to 12 hours, when it is still possible to correct deficits by the administration of oral colostrum or plasma.

Single radial immunodiffusion (SRID)

- This test is most commonly performed in a laboratory although test kits are available.
- It takes 15–24 hours for completion and thus is not used for routine assessment of serum IgG. It is however very accurate and can be used as a confirmation test and for quality control if other tests are routinely used.

Zinc sulfate turbidity test (Fig 3.51)

- Commercial kits are available or can be prepared "in house."
- It is quick, cheap and accurate, although accuracy is decreased in lower ranges (<4 g/L).
- Hemolysis can give falsely elevated results.
- Serum should be used instead of plasma as elevated plasma fibrinogen may result in inaccurate results. Serum is slower to obtain than plasma and thus makes this test unsuitable if rapid testing is required.

Method

1. 250 mg of hydrated zinc sulfate is added to 1 L of freshly boiled water.
2. 6 mL aliquots of the warm solution are placed into sealed 7–10 mL plain vacuumed collection tubes. These tubes remain useful for several months. When used the tube should be clear. Any cloudiness before the test commences indicates CO_2 absorption into the solution.
3. Add 0.1 mL of serum.
4. Mix.
5. Wait 10 min for a qualitative result (turbidity indicates a positive result, i.e. adequate immunoglobulin content). Wait 60 min for a quantitative result (measure against calibrated barium sulphate standards)

Latex agglutination

- This test involves specific equine immunoglobulin (IgG) antiserum which is coated onto latex reagent and mixed with the sample being tested.
- Commercial kits are available. Whole blood, plasma or serum can be used depending on the kit.
- Positive agglutination occurs within 15 minutes and results can be quantified.
- The test is simple and rapid and can be performed stable-side, although kits may be expensive.
- The test is not affected by hemolysis but can be adversely affected by temperature.
- There is a high accuracy at or below 4 g/L but above this it may be poor.

Concentration immunoassay technology test (CITE test)

- A variety of commercial kits are available. They all require accurate pipetting and instructions provided with the kit should be followed exactly.
- Whole blood, serum or plasma can be used.
- The test uses a color spot with calibration standards that correlate to concentrations of 2, 4 and 8 g/L of IgG.
- The test is rapid (10–15 minutes) and results correlate well with SRID results.

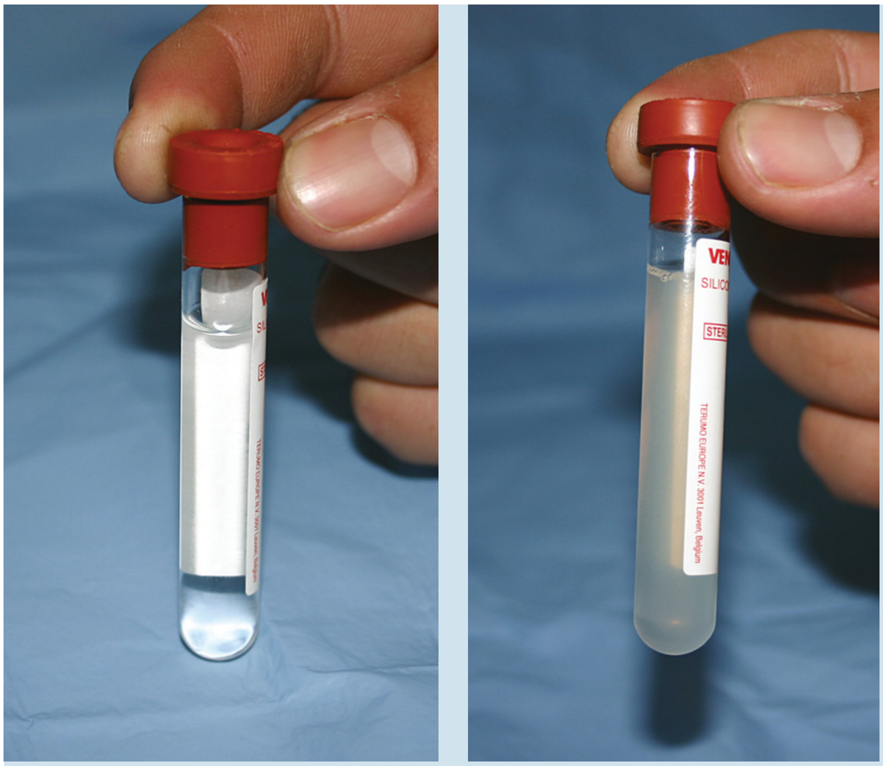


Figure 3.51: Zinc sulphate turbidity test. The zinc sulphate solution is clear before the addition of serum and becomes cloudy after the addition of the serum, indicating the presence of immunoglobulin.

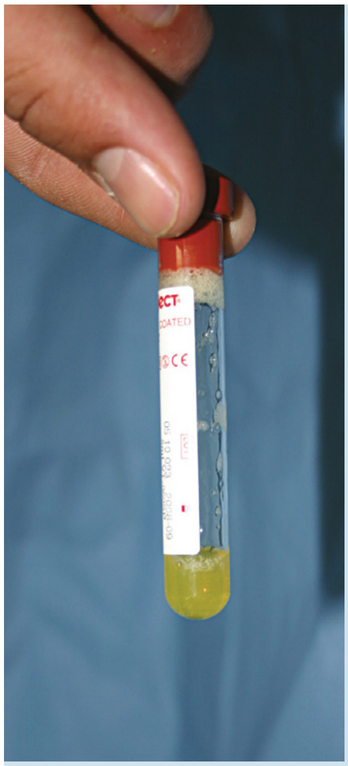


Figure 3.52: Glutaraldehyde test. Note the firm clot that is formed after the addition of serum to the glutaraldehyde.

Glutaraldehyde coagulation (Fig 3.52)

- Chemical grade 25% glutaraldehyde is used to make the 10% glutaraldehyde solution for testing. This forms an insoluble precipitate when mixed with basic proteins.
- Commercial kits are available or it can be prepared in-house.
- It is inexpensive and reliable but glutaraldehyde is a hazardous substance.
- Hemolysis can result in overestimated IgG.
- This test should be periodically quality checked against the SRID test.

Method

1. Prepare a 10% solution of glutaraldehyde with deionized water.
2. Add 0.1 mL of the glutaraldehyde solution to 1 mL of serum and start the clock.
3. Measure the time for clotting of serum to develop.
4. Clotting in <10 min = IgG > 8 g/L
5. Clotting in 60 min = IgG 4–8 g/L

Total serum protein

- This is an unreliable indicator of globulin status because of the wide range of normal concentrations.
- The correlation of total serum protein to IgG levels is also inaccurate in the diseased foal (clinical and subclinical) due to the increase in other globulins (alpha and beta globulins and acute phase proteins).

Electrophoresis

- Electrophoretic determination of individual plasma proteins is a very accurate test.
- It is time consuming and requires specialized equipment but is useful as a quality control test.

Blood tests: complete blood count and serum biochemistry

Appendices 3 and 4 outline normal values for routine blood tests.

Care of the sick neonate, diagnostics, procedures and nursing care

A foal may be obviously ill from birth, for example those that have required resuscitation after delivery or did not rise and nurse by 3 hours; or may appear normal after delivery and develop signs of illness (failure to nurse, depression, seizures) within 72 hours. The following pages describe veterinary interventions from resuscitation of the newborn, diagnostic tests and clinical procedures to nursing care.

Resuscitation (Figs 3.53–3.60)

Cardiopulmonary resuscitation should be performed in newborn foals that have any of the following:

- absent or gasping breathing
- absent or irregular heartbeats
- heart rate <40 BPM
- non-responsive
- delivered by C-section.

Resuscitation efforts frequently fail due to a lack of preparation both on the part of the farm staff and veterinarian. Farm staff/owners

Figure 3.53: Neonatal resuscitation kit. Having all possibly needed medications pre-drawn in syringes saves time and spares hands during a resuscitation effort.

Figure 3.54: Intensive care bed and equipment ready to receive foal following resuscitation.

Figure 3.55: Self inflating valve-bag suitable for assisted ventilation during CPR.

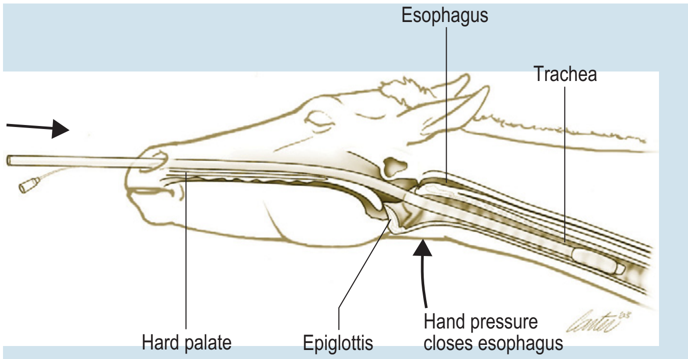


Figure 3.56: This illustration demonstrates the correct head positioning for nasotracheal intubation (from Fielding CL, Magdesian KG 2003 Cardiopulmonary cerebral resuscitation in neonatal foals. Clinical Techniques in Equine Practice 2(1):11, Figure 1).

Figure 3.57: Towel drying a foal delivered by C-section in a sternal position to stimulate respiration.

Figure 3.58: Ambubag being used to ventilate a foal with room air after being delivered by terminal C-section. This foal had an HR >60 BPM with a mean BP >60 mmHg but required ambubag ventilation for 40 minutes before spontaneous respiration occurred. Thereafter the foal made an uneventful recovery and went home 3 days later.

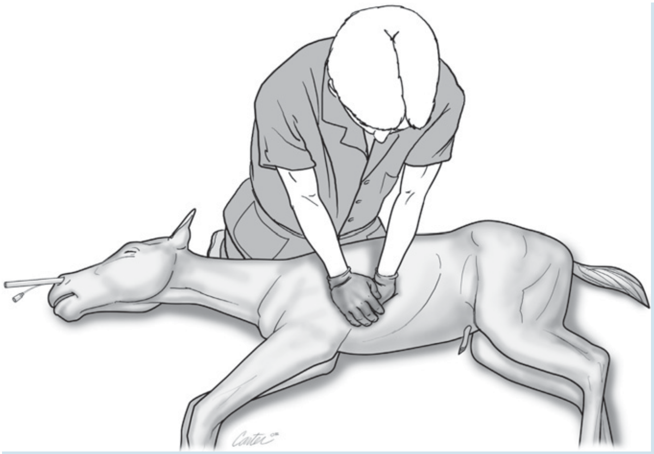


Figure 3.59: Illustration demonstrating proper positioning for the performance of effective external chest compressions (from: Fielding CL, Magdesian KG 2003 Cardiopulmonary cerebral resuscitation in neonatal foals. Clinical Techniques in Equine Practice 2(1):14, Figure 4).

should be educated about the need to contact the veterinarian as early as possible, e.g. if delivery is prolonged. Similarly veterinarians should have a resuscitation kit at hand at all times with necessary drugs pre-loaded in syringes and clearly labeled. Resuscitation is a team effort and those attending foal deliveries at the farm level should be educated in advance about their role in the case of a resuscitation.

The ABC of resuscitation

Following delivery the foal's airway should be cleared and the animal should be vigorously dried. If a regular heart rate (60–120 BPM) and

Figure 3.60: This image demonstrates the correct sites for placement of defibrillator paddles in the foal.

regular spontaneous breathing are achieved then further intervention is not required.

A – Airway

- The airway should be cleared using a 60 mL syringe and rubber tubing. Suctioning should only be performed if meconium is present and then only for less than 10 seconds as it can cause bradycardia and cardiac arrest via vagal reflexes.
- Postural drainage can help drain fluid from the lungs. It can be achieved by placing the foal's head at a level below the shoulder. ***The hindquarters should not be raised and the foal should not be inverted as this may actually inhibit respiration.***
- Vigorous drying not only provides tactile stimulation but also helps to minimize heat loss and oxygen consumption. Radiant heat should be provided following drying.

B – Breathing

- Foals that are not breathing, have gasping breaths or a heart rate <60 BPM require artificial respiration. A nasotracheal tube and self-inflating resuscitation bag should be used. Intubation can be nasotracheal or endotracheal and placement should be confirmed before bagging commences.
- Disconnecting the resuscitation bag and checking for a regular respiratory pattern and a respiratory rate >16 BPM will assess spontaneous ventilation. If adequate and the heart rate is >80 BPM, ventilation can be stopped. The tube should be left in place for a short time in case ventilation has to be re-started.
- The respiratory stimulant doxapram (0.5 mg/kg IV) may be used for resuscitation but there is some controversy about its use because it has been shown to decrease cerebral blood flow and increase myocardial oxygen consumption.

C – Circulation

- Thoracic compression should be initiated if the heart rate remains undetectable following the initial 30 seconds of positive pressure

ventilation or if there has been a poor response to initial drug therapy.
- Many authors advocate initiating thoracic compression if the heart rate is <40 BPM, however it is the experience of the editors that drug therapy provides better results than thoracic compressions in bradycardic foals.
- To perform thoracic compressions the foal should be placed on a firm flat surface with a firm support below the sternum and the ribs should be palpated for fractures.
- The person performing the compressions should kneel behind the foal. The heel of one hand is placed at the level of the foal's shoulder behind the triceps mass and the other hand is placed over the first.
- One breath should be given for every 5 thoracic compressions (TC) and CPR should not be stopped for greater than 10 seconds to assess circulation.

D – Drug therapy

- A heart rate <40BPM is an indication for drug therapy. However, older foals that present requiring resuscitation should have a blood glucose measurement performed to rule out hypoglycemia as a cause of bradycardia.
- Glycopyrrolate (0.2 mg/mL) can be administered initially (use as a 1× dose of 1 cc per 50 kg IV). This anticholinergic reduces muscarinic symptoms such as bradycardia. This treatment can also be combined with epinephrine but if doing so use half the normal dose of epinephrine initially (0.25–0.75 mL of the 1 : 1000 solution for a 50 kg foal).
- Epinephrine at 0.01–0.03 mg/kg (0.5–1.5 mL of the 1 : 1000 solution for a 50 kg foal) should be administered every 3–5 minutes until spontaneous circulation is achieved. It is important to continue ventilation if needed following administration of the drug. Thoracic compressions should be initiated/continued if the foal remains bradycardic after drug therapy.
- The administration of polyionic fluids for volume expansion is recommended in foals that have weak pulses with a good heart rate, remain pale or cyanotic after oxygenation or have poor response to resuscitative efforts.
- Defibrillation is indicated for foals with ventricular fibrillation or pulseless ventricular tachycardia. The first defibrillation should consist of up to three consecutive discharges: 2 J/kg followed by 4 J/kg followed by 4 J/kg. All subsequent defibrillations should be at 4 J/kg and should take place approximately 1 minute after treatment with epinephrine or an anti-arrhythmic drug.

E – Effectiveness

- In the absence of a detectable heartbeat the effectiveness of CPR can be assessed by pupillary constriction. The pupillary light reflex (PLR) is present at birth although slower than normal. The use of a penlight to assess PLRs during resuscitation is useful. A widely dilated non-responsive pupil indicates inadequate resuscitation and a poor prognosis.
- A capnograph can also be used to determine end tidal CO_2. Values >15 mmHg and <50 mmHg indicate good perfusion and a better prognosis. Values <10 mmHg indicate a poor prognosis as there is less CO_2 being transported to the lungs.
- When to stop resuscitation efforts is unclear. In the absence of both spontaneous circulation and respiration a time frame of 10 minutes is probably realistic. However the presence of a regular heartbeat (>60 BPM) in the absence of respiration warrants a prolonged effort.

Protocol for assessment and procedures in sick neonates not requiring resuscitation

- Perform observational assessment of foal with particular attention to level of alertness, respiratory rate and pattern and maturity.
- Place the foal in a suitable warm and safe environment (if this has not already been done by farm staff). Ideally the foal should be weighed at this point. In neonates this can simply be done by getting the person carrying the foal to stand on bathroom scales, and deduce the foal's weight by subtraction of the person's weight.
- Assess mucous membrane color and CRT. If available and necessary, use oxygen throughout the remainder of the examination. If there is no immediately evident requirement then it is best to wait until after oxygen requirements have been determined.
- Check the rectal temperature. This can be performed by an assistant or a member of the farm staff. (Oxygen saturation assessment using pulse oximetry could be performed immediately after or before rectal temperature assessment.)
- Clip and aseptically prepare a site for obtaining a blood culture and catheter placement.
- Obtain a blood culture.
- Place an IV catheter.
- Draw blood samples for CBC, rapid glucose determination (heparinized) and serum biochemistry (serum electrolytes, creatinine and IgG are the initial requirements unless there is an indication for other tests, e.g. icterus noted during mucous membrane examination would indicate measurement of bilirubin). This can be done at the same time as the blood culture is obtained if there is an assistant at hand.
- Measure blood glucose using a glucometer. If < 80 mg/dL start 5–10% glucose solution.
- If not hypoglycemic start pre-warmed polyionic fluids such as Normosol^{R} or 2.5% glucose in 0.45% saline.
- If the foal is not nursing place a feeding tube.
- Perform standard nursing care procedures. (Chlorhexidine solution to navel, tetanus prophylaxis if the mare was not vaccinated 2–4 weeks prior to foaling, enema administration etc.)

If the foal does not respond appropriately to the steps above or if the foal is presented recumbent or hypotensive; if septicemia is suspected or if the foal shows signs of unreadiness for birth, additional tests (listed below) should be performed without delay. At this point the decision may be made to refer the foal.

- Blood pressure measurement – a tail cuff can be tried initially but many hypotensive foals will fail to register a reading, and assessment using a manometer will be required.
- Blood gas measurement.
- Thoracic radiography/ultrasonography.
- Abdominal ultrasonography to assess gut motility is also useful.
- Antimicrobial selection (see Appendix 5).

Whether or not to refer

The decision to refer a foal is not always an easy one and should be based on the following.

Available equipment, nursing help and clinical expertise

- If the correct equipment such as intravenous catheters, urinary catheters, feeding tubes, oxygen supply and proper facilities are not available then refer immediately.
- While help at farm level is often enthusiastic, bear in mind that many sick foals need prolonged care which can become tiresome and boring for farm staff. In addition long hours spent attending a sick foal may lead to neglect of other foals. "Down" foals require specialized nursing care. A trained nurse may notice a subtle but very significant change in clinical condition that could go unnoticed at farm level and adversely affect the outcome of the case.
- Foal care is a specialization. Even if a foal is not referred for financial or other reasons, always consult with a specialist. Most will be delighted to help either by visiting the farm or via phone consults.

The rate at which the foal's clinical condition has deteriorated

- A foal whose clinical condition has deteriorated quickly will not benefit from the "wait and see" option. However, a foal that is likely to be dead on arrival should not be referred.

Value of the foal (emotional or commercial)

- It is also important to consider the whole foal when making the decision to refer and not just the immediate clinical condition of the foal. Foal intensive care is expensive. Most foals will have a one-week hospital stay at an average cost of $1000 per day (at 2007 rates). The cost and potential outcome should be discussed carefully with the client. The owner should be aware of the fact that referral does not necessarily guarantee a successful outcome.

The journey involved

- Will the foal survive a lengthy journey?
- Does the foal need stabilization before transportation or treatments during the journey?
- What type of vehicle is available for the journey? Will it allow separation of the mare and foal if necessary for treatment? Horse boxes/floats are the best as they allow easier access to the foal for treatment, have better temperature control and more space which may be important for the mare. Foals should not be transported in the boot of cars or the back of pickup trucks.

Other transportation considerations

- Load the foal first within sight of the mare.
- Separate the mare and foal if the foal is not ambulatory and coordinated.
- Provide adequate padding and warmth. Carpeting on the floor of the box helps to prevent slipping and improves insulation. Straw bedding should be used as other forms of bedding such as shavings can be ingested, inhaled or cause irritation of the eyes.
- Foals which are down should be placed on a blanket or rug.
- Rectal temperature should be taken prior to the journey – if below normal, measures should be taken to warm the foal. These should include blankets and warm fluids but direct heat to the body such as electrically heated pads or hot water bottles should be avoided as they may cause tissue necrosis.
- Fluid therapy is required in foals that are dehydrated. Where possible blood glucose should be measured prior to departure and added to fluids if required.
- Feeding will also be required if the journey is likely to be more than 2 hours in duration. If the foal is able to nurse, the journey should be halted to allow the foal to do so. If the foal is not able to nurse then a feeding tube should be placed prior to traveling.

Other aspects of referral

- Call the referral center before the foal is sent.
  - Always let the referral center know that you are coming. Showing up unannounced is not only impolite but is not the best thing for the foal as it does not allow time to "set-up" for the new arrival. In addition, intensive care units fill quickly during the breeding season and even the largest facilities may run short of space and direct a client to another facility.
  - Calling in advance will also allow early discussion of the case and any proposed treatments prior to arrival; e.g. most facilities will prefer that foals have not received antibiotics so as not to interfere with blood culture results.
- Providing any available hematology or serum biochemistry results that are available over the phone may not only help guide early treatment but may assist in forming an early list of differentials. For example, a foal with a history of stranguria and a high serum potassium and creatinine would be highly suspicious of a ruptured bladder and would allow forewarning of both medical and surgical teams. If such results are provided over the phone always send copies with the foal or fax them also.
- Send a written account of the case with the foal. This should include history with copies of the initial examination form and foaling form, any procedures that have been undertaken and their results, e.g. ultrasound findings, laboratory results if available, insurance information and relevant contact numbers and addresses.
- Any samples taken should be packaged correctly and sent with the foal. The placenta should also be sent if available and should be packaged in a *clean* plastic bag. Unless the journey is very short the placenta should be packed on ice.
- If the mare is not traveling with the foal, some milk/colostrum should be obtained and sent with the foal.

Diagnostics

Many diagnostic tests are covered under their respective chapters; the remainder are described below.

Blood pressure measurement

Blood pressure monitoring is essential in intensive care patients and may be measured indirectly or directly. Therapy is based on mean rather than systolic blood pressure as mean pressure is a better

predictor of organ perfusion. Mean pressures should be maintained above 60 mmHg with normal mean BP being 70–100 mmHg in neonatal foals.

Indirect

- Most indirect measurements involve the use of a cuff most commonly placed around the coccygeal artery. Improved technology has resulted in more reliable results with this method; however, two readings should always be taken and any reading where the stated pulse rate does not match that measured manually should be disregarded.
- The position of the foal should also be taken into account as readings will be lower in foals that are recumbent.
- Accurate readings may be difficult to obtain in dehydrated foals or in foals that actually have low blood pressure.

Direct

- Direct measurement of blood pressure involves the placement and where necessary maintenance of intra-arterial catheter. Indwelling catheters allow for continuous measurements to be taken.

Other hemodynamic measurements

Central venous pressure

- In young foals, placement of a 20 cm or 30 cm-long IV catheter into a jugular vein allows intraluminal pressure in the cranial vena cava to be measured. Luminal hydrostatic pressure in the vena cava reflects venous return; that variable closely or identically approximates right atrial pressure, which reflects ventricular preload.
- Indications for measurement of CVP include conditions that affect intravascular volume such as dehydration, renal dysfunction and severe hemorrhage. Other conditions that require careful fluid administration such as cerebral edema and increased intracranial pressure are indications for CVP measurement.
- Water manometers are commonly used to measure CVP. The baseline measurement is assessed at the level of the left atrium (ideally the patient is either standing or sternal).
  - Normal is usually in the range 10–12 cm H_2O but is variable and can be lower in normal foals.
  - Decreased CVP indicates decreased venous return and suggests inadequate intravascular fluid volume.

Without knowing baseline CVP values of an individual foal in a clinical situation, a CVP value that is within reference range is difficult to interpret because hypovolemia may be present in the face of an apparently normal CVP. Therefore as clinicians we must ***look for "trends" in the CVP results. If the CVP has been steadily rising on serial readings then the patient may be in a hypervolemic state requiring decreased fluid therapy. The opposite is true if the CVP is noted to be steadily declining.***

Assessment of hypoxemia and determining oxygen requirement (Figs 3.61–3.65)

Hypoxemia (PaO_2 <70 mmHg) can have many causes in newborns including birth asphyxia, congenital heart disease, atelectasis, pneumonia and hypoventilation. It can be seen in older neonates in association with septicemia, unreadiness for birth, hypoxic ischemic encephalopathy (HIE) and in any foal that has been recumbent for prolonged periods. Hypoxia may be difficult to detect by clinical examination alone and specific procedures are often required.

Subjective assessment

- History and clinical signs. Signs of asphyxia, apnea or labored respiration. Pale or cyanotic mucous membranes – although it should be noted that cyanosis is not readily apparent until PaO_2 is <40 mmHg.
- Auscultation and percussion abnormalities of the chest. This is unreliable as there is poor correlation between abnormalities and the severity of disease.

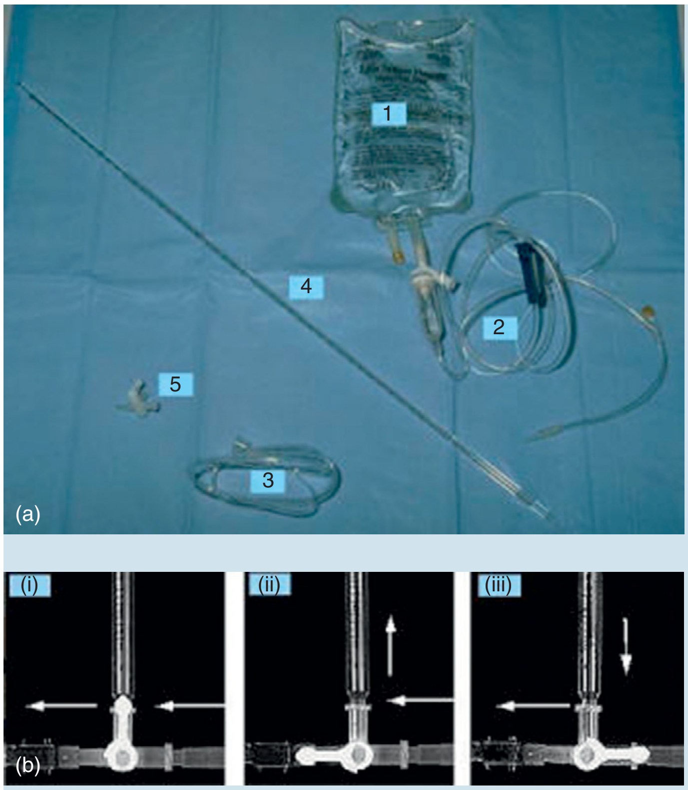


Figure 3.61: (a) The equipment needed for measurement of central venous pressure includes a sterile bag or bottle of fluids (1), with attached fluid administration set (2), an IV extension set (3), a manometer (4) and a stopcock (5). (b) In the images above, the foal is on your left and the bag of fluids is on your right. The direction the white knob is pointed is the OFF position of the stopcock. The white arrows indicate the direction of fluid flow. Initially the white knob is turned straight up towards the manometer, allowing fluid to flow from the fluid bag to the patient's catheter to assure the catheter is patent (i). If fluid does not flow freely into the patient's catheter a valid CVP reading will not be obtained. Then the knob is turned toward the patient (ii) and fluid will fill the manometer. The manometer should not contain any air bubbles. If air is present in the manometer or fluid line, let the fluids run, overfilling the manometer until all air is purged from the system. Then turn the knob toward the fluids (iii). The level of fluid in the manometer will fall (the fluid is running into the patient's catheter) until the height of the fluid column exerts a pressure equivalent to the patient's CVP. The top of the fluid column will slightly oscillate up and down as the animals' heart beats and as the animal breathes.

Figure 3.62: Pale and cyanotic mucous membranes in a foal after acute hemolysis. Cyanosis is a very subjective assessment of hypoxemia.

Figure 3.63: Blood gas collection from the metatarsal artery in a neonate. A small diaper dampened with water and placed in a microwave for 20 seconds was placed over the artery for 30 seconds before collection. The warm compress allowed for dilation and easy visualization of the artery. Topical application of a local anesthetic cream may also decrease movement during collection.

Objective assessments

Blood gas analysis

- Arterial samples:
  - Arterial blood gas (ABG) analysis should be performed on any neonate patient with respiratory compromise. Arterial samples can be obtained from the tarsal, brachial or median arteries. Alternative sites include the transverse facial behind the eye, the facial artery, the femoral artery or the carotid artery. The umbilical artery can also be used in foals less than 6 hours old but the risk of infection is increased at this site.
  - The site should be aseptically prepared and clipped to facilitate visualization and palpation of the artery. Full aseptic precautions should always be taken. Failure to do so can result in septic thrombi being deposited in distal capillary beds.
  - Either a topical anesthetic like EMLA Cream® (AstraZeneca, London, UK – lidocaine and prilocaine) or a small bleb of lidocaine placed subcutaneously may decrease motion in response to sample collection.

Figure 3.64: Pulse oximetry being performed on the preputial skin.

Figure 3.65: Foal with nasogastric and oxygen tubing in place. The nasogastric tube is sutured to the muzzle and the O_2 tubing is taped to the nasogastric tube. If O_2 tubing alone is being used then this should be sutured directly to the muzzle.

- The blood should be collected in a heparin coated syringe, evacuated of air bubbles and sealed with a rubber stopper. The sample should be analyzed within 10 minutes if at room temperature or 3 hours if placed on ice.
- PaO_2 measurements assess the pulmonary oxygenating capability. Oxygen tension in arterial blood below 80 mmHg is associated with hypoxemia and arterial saturation of <95% and indicates a requirement for oxygen therapy.
- Values below 60 mmHg indicate severe hypoxemia and warrant careful monitoring as assisted ventilation may be required.
- PaO_2 measurements can vary from normal in the early stages of life and when collected in lateral recumbency. Clinically normal neonatal foals can have PaO_2 below 80 mmHg in the first 12–24 hours of life. Immediately after birth foals can have a PaO_2 between 40 and 50 mmHg and if in lateral recumbency the PaO_2 can be 39.7 ± 2.1 mmHg.
- $PaCO_2$ measurements in arterial blood assess the ventilatory status of the patient. Normal foals' carbon dioxide tension in arterial blood ranges between 42 and 45 mmHg.
- $PaCO_2$ >60 mmHg is associated with hypoventilation and may warrant chemical stimulation of ventilation or mechanical ventilation.
- Struggling may result in decreased $PaCO_2$ and a more alkaline pH.
- If an unexpectedly low PaO_2 is obtained a venous sample should be submitted in comparison, as a venous sample may have been obtained and believed to be an arterial sample.

- Venous samples:
  - These are of limited value for O_2 assessment but are important for acid–base assessment.

Pulse oximetry

Many monitors that measure blood pressure will also allow for concurrent measurement of oxygen saturation of arterial hemoglobin. Such measurements are useful especially in foals with respiratory compromise as they allow assessment of both the efficacy of therapy and the clinical condition. However, the transducer and site used may affect results. The most common sites are the rectum, vulva or prepuce. Measurements should be >90% with lower levels indicating a requirement for oxygen therapy.

Oxygen therapy (Fig 3.66)

Figure 3.66: Oxygen humidifier.

- Place the foal in a sternal position, unless there is some other condition, e.g. fractured ribs, which precludes such positioning.
- Face mask.
  - High inspired oxygen concentrations can be obtained.
  - Dead space within the mask can be reduced by ensuring that the patient's nose fills the mask as much as possible.
  - Leaks allow room air to be drawn into the mask during inspiration. This is permissible as long as the inspired oxygen concentration is still sufficient to alleviate the hypoxia. If leaks are significant the oxygen flow rate may be increased or an airtight seal could be formed around the muzzle, in which case expired gases would have to be eliminated via exhaust or CO_2 absorber.
  - Face masks are usually not as well tolerated as insufflation catheters.
- Oxygen insufflation catheter.
  - These may be placed in the nasopharynx or intratracheally. Measurement and marking of a length of tubing from the nostril to the medial canthus of the eye will allow proper positioning in the nasopharynx. Tubing placed in the nasopharynx should be sutured at the nostril to prevent displacement.
  - Several holes should be present near the end of the catheter tip to facilitate diffusion of oxygen and prevent jetting against a single area of epithelium.
- The supplied oxygen should be humified by bubbling through water at room temperature prior to reaching the patient.
- If altered positioning and oxygen therapy do not improve the hypoxemia then endotracheal intubation and positive pressure ventilation are indicated.
- Hypoxemia with hypercapnia (>60 mmHg) will not be corrected by oxygen therapy alone. The foal's ventilation must be improved. If $PaCO_2 > 65$ mmHg and rising and pH < 7.2 mechanical ventilation may be required. If mechanical ventilation is not possible respiratory stimulants such as caffeine or dopram continuous rate infusion (CRI) can be used to treat abnormally slow breathing patterns associated with central depression of the respiratory center. Alternatively intermittent intubation and ambubag positive pressure ventilation 2–4 times daily can be tried to expand collapsed alveoli.

Rapid blood glucose measurement (Fig 3.67)

- Blood glucose measurement is critical in neonatal foals, especially in those on parenteral nutrition. Wet chemistry methods are the most reliable but as specialized equipment is needed, results are not immediately available.

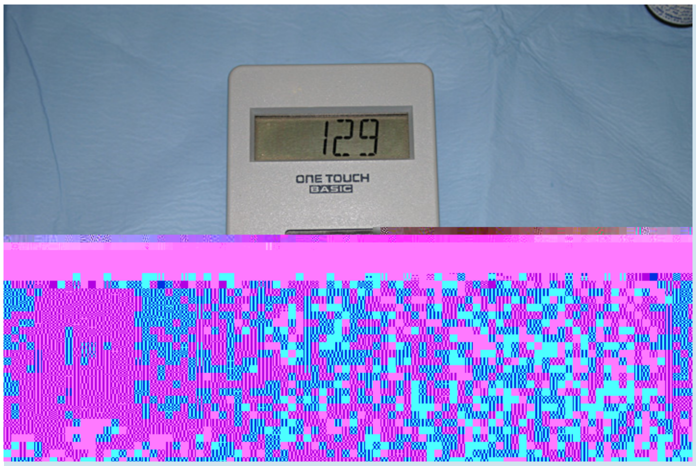


Figure 3.67: Glucose meter for rapid blood glucose determination.

Figure 3.68: Biphasic blood culture. The specified sample volume is taken under strict aseptic conditions and then added to the collection bottle which is submitted to the laboratory. See text for description of technique.

- Rapid measurements can be obtained by using analyzers available for humans (used by diabetics). Although calibrated for humans, they are generally reliable for foals, especially if regular quality assurance is performed.

Blood culture (Fig 3.68)

- Blood cultures are extremely useful for diagnosing and guiding treatment of septicemic foals and are also useful in monitoring nosocomial infections.
- Blood cultures are best performed prior to antibiotic administration and as early in the disease process as possible and as such should be taken by the first attending veterinarian.
- Aseptic preparation of the site and direct withdrawal of the blood from the selected (usually jugular) vein is required to decrease contamination. After withdrawal of the blood a new needle should be placed on the syringe before the sample is injected through the injection port (which has been wiped with an alcohol swab) into the culture bottle. The person handling the blood culture bottle should also be wearing sterile gloves.
- False positives and false negatives are possible but the percentage of performed tests that provide invaluable information is high.

Clinical procedures

Intravenous catheter selection and placement (Figs 3.69–3.75)

Intravenous catheter placement:

- is required for:
  - continued administration of intravenous antibiotics
  - intravenous fluid therapy
  - parenteral nutrition
  - plasma/blood administration
- should also be used in foals receiving one-off doses of irritant antibiotics, e.g. intravenous administration of oxytetracycline as a treatment for tendon contracture.

In all except the latter the foal is by definition ill and thus aseptic preparation and placement is essential.

Selection

- The type of catheter selected will depend on the anticipated length of treatment and the items to be administered. However, treatment time is often underestimated and the need for parenteral nutrition is often not apparent until after catheter placement. Placing the best available catheter early on frequently proves to be the most economical option as it decreases the frequency of catheter changes and decreases complications such as thrombophlebitis.
- 16G catheters are generally used but 18G catheters can be used for 1× daily antibiotic administration.
- Longer catheters are also generally better; $5^{1}/_{4}''$ should be used for jugular placement, $3^{1}/_{4}''$ can be used for a single jugular treatment or when peripheral placement is required, e.g. cephalic vein.

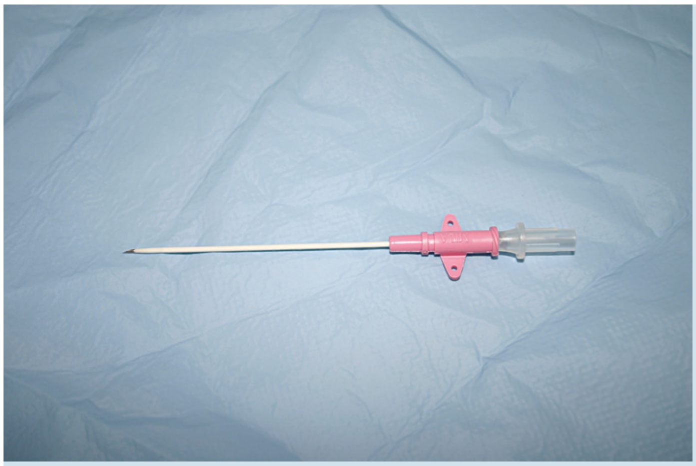


Figure 3.69: 18G $3^{1}/_{2}$″ MILA polyurethane catheter. This catheter is suitable for short-term use (up to 72 hours).

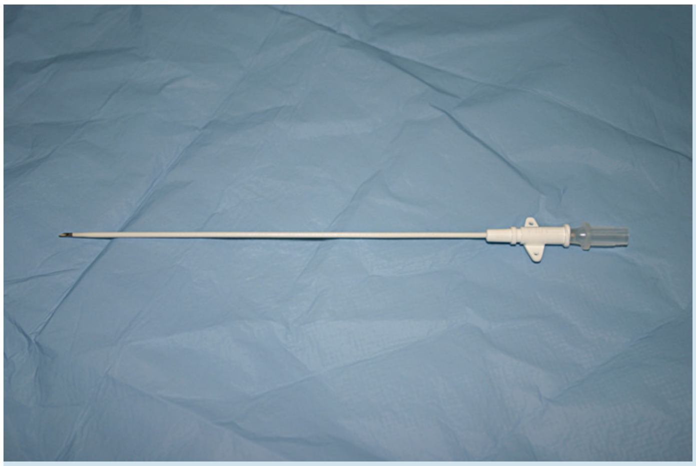


Figure 3.70: 16G $5^{1}/_{4}$″ MILA polyurethane catheter suitable for jugular placement and the administration of fluids. Again this catheter is suitable for use up to 72 hours.

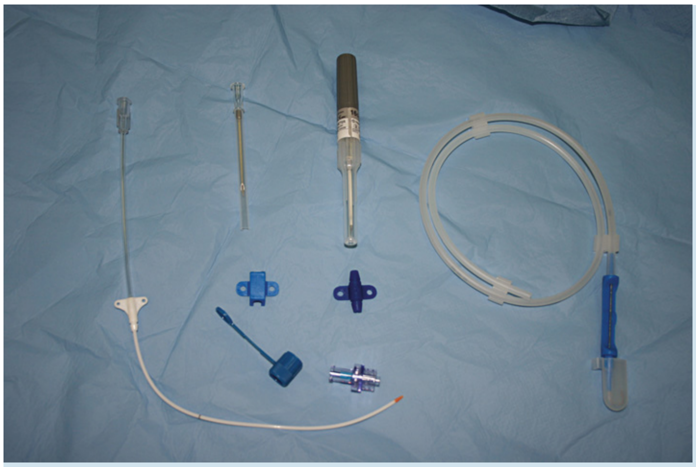


Figure 3.71: MILA 16G 15 cm long-term "over the wire" catheter.

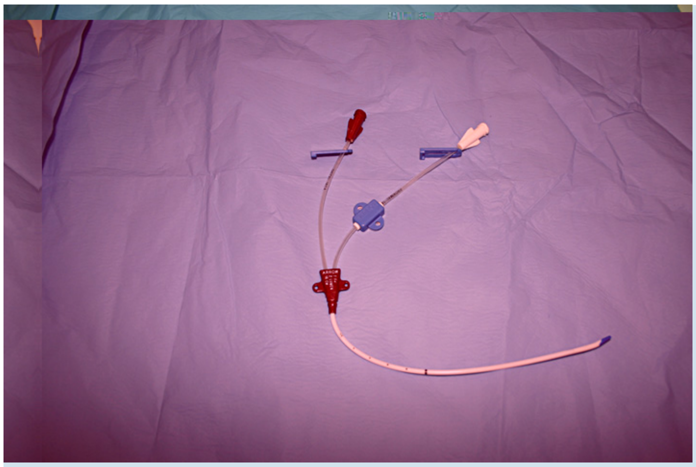


Figure 3.72: Double lumen ARROW catheter.

- Teflon catheters are the cheapest and the most easily placed but are highly thrombogenic.
- Polyurethane catheters are the least thrombogenic but the most difficult to place, with many requiring an "over the wire" insertion.
- Many catheters designed for long-term use also have built in T-ports. Other catheters should have T-ports attached after placement to avoid manipulation of the catheter itself. Kinking most often occurs where the catheter enters the skin.
- Multiple lumen and antibiotic impregnated catheters are also available.
- Catheters designed for long-term use may be left in place for several weeks provided there is no evidence of inflammation. Other catheters should be changed q72 hours, or sooner if swelling or inflammation are apparent.

Other sites for catheter placement

- Cephalic or lateral thoracic veins can be used for antibiotic and small volume fluid administration. These sites are not generally suitable for the delivery of large volumes of IV fluids or the administration of parenteral nutrition.
- Arterial catheters can be placed in the facial, femoral or greater metatarsal arteries.

Complications (Fig 3.75)

- Hematoma formation or excessive bleeding at the site of insertion may reflect poor placement technique or a clotting abnormality.
- Air embolization. If the cap of a catheter is dislodged then air may be drawn in resulting in an air embolus which could, in severe cases, be fatal.
- Extravascular leakage either reflects poor catheter placement or a damaged catheter.
- Kinking usually reflects poor placement technique or excessive manipulation of the catheter. Kinking of the catheter decreases lumen size and thus affects fluid flow rates. If a flow rate does not return to normal after flushing with a heparin solution then kinking should be suspected and the catheter should be removed.
- Thrombophlebitis. This can present as localized abscessation but more often as hardening of the vein. If a catheter is not removed at the earliest signs of inflammation then the process continues, resulting in a cord-like vein and in some cases occlusion of the vessel. Veins which have become hardened in this way cannot be re-used for venous access. Thrombophlebitis does not always reflect

(a) (b) (c) (d) (e) (f)

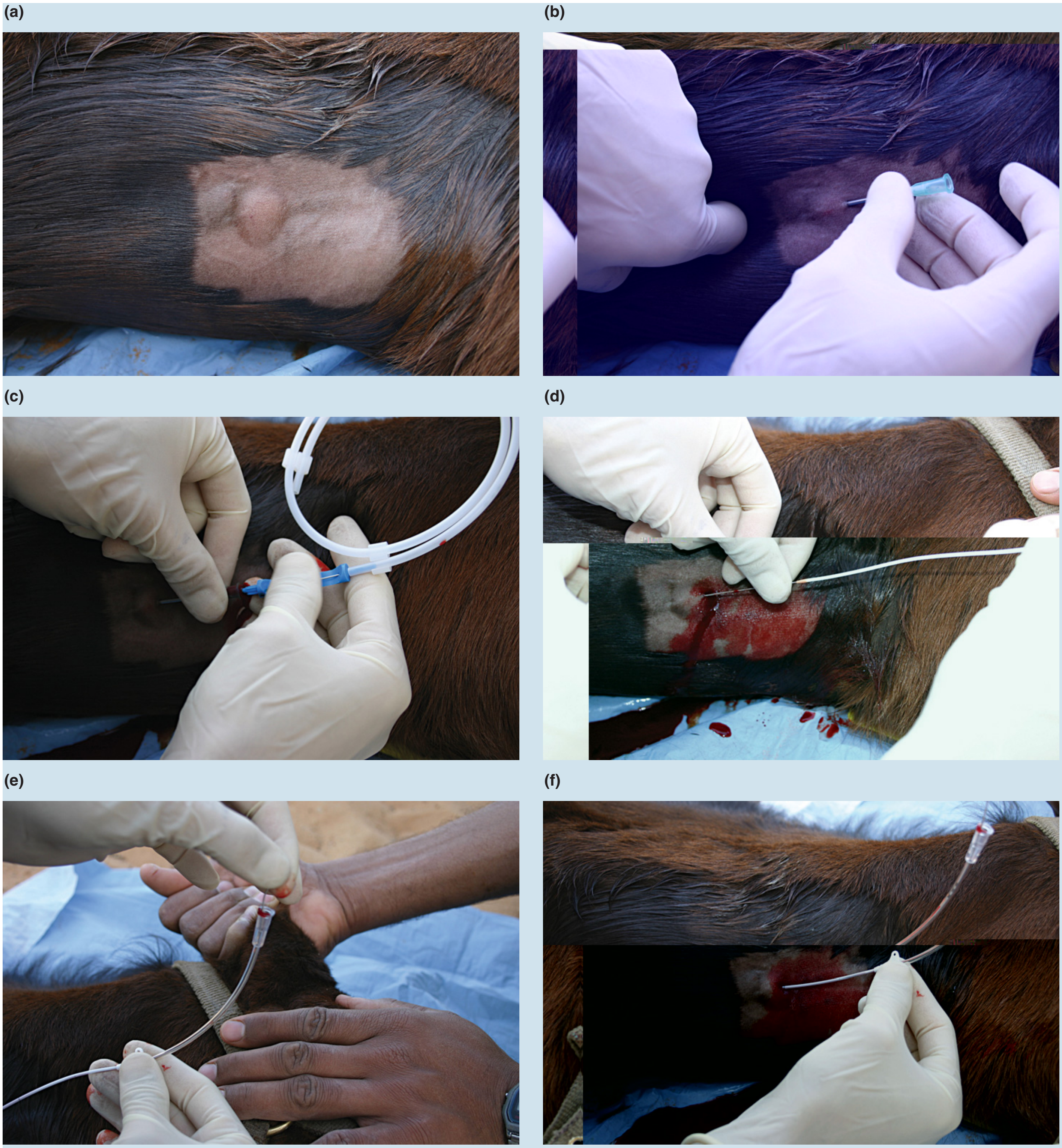


Figure 3.73: Placement of an "over the wire" catheter. (a) The site is surgically prepped. A bleb of local anesthetic is then placed at the site. (b) A 14G needle is inserted into the vein. (c) The J-wire is then advanced through the needle. (d) The needle is then removed over the wire. (e) The catheter is then advanced over the tip of the wire. The wire is then fed up through the catheter until it can be grasped at the plastic hub of the catheter. (f) The catheter is then passed through the skin and advanced down the vein. It is of utmost importance not to let go of the J-wire at any stage.

Figure 3.74: Placement of an "over the needle" catheter. Again the site is surgically prepared and a bleb of local anesthetic is placed at the site. (a) Some operators prefer to pre-drill the skin site depending on the type of catheter being used and the hydration status of the patient. Passage through the skin creates a lot of drag and in many cases is the cause of the catheter kinking during placement. If pre-drilling is to be performed the skin should be lifted up and the needle should only be passed through the skin, not into the vessel as this will cause localized hemorrhage. (b) The vein is then raised and the catheter and stylet with bevel up are passed through the skin into the vessel. (c, d, e) As soon as a blood flashback is seen the catheter is advanced while holding the stylet still. Advancing the stylet any further after the flashback has been seen is likely to result in vessel trauma. (f) The stylet is then removed and the correct positioning of the catheter is verified by either aspirating with a syringe or occluding the vessel below the end of the catheter until blood is seen to emerge from the hub. (g) A T-port is then attached to the catheter and both the T-port and catheter are sutured to the skin.

Figure 3.75: 8-month-old filly with mitral regurgitation secondary to endocarditis which was thought to have resulted from thrombophlebitis which occurred during hospitalization as a neonate. Note the small size of this filly. This filly also lost her distal tail as a result of a urinary catheter that was too tightly taped to the tail.

poor technique but in many cases is a result of a hypercoagulable state in septicemic patients. Occasionally endocarditis or organ thromboembolization can occur secondary to thrombophlebitis, resulting in serious sequelae.

Figure 3.76: Urinary catheters: the catheter on the left is 30 cm and suitable for use in neonatal fillies, the catheter on the right is 55 cm and suitable for use in neonatal colts. Both are 12F.

Bladder catheterization (Figs 3.76–3.78)

Placement of a urinary catheter

- The skin site of entry should be aseptically prepared and sterile gloves worn. Lubricant, collection system (if used) and suture materials to be used for affixing the catheter to the skin, or tape for attaching the catheter to the tail, should be within easy reach.
- The collection system should remain intact and closed once placed.
- Catheters with a balloon reservoir at the tip should be used, and for most light-horse foals, catheters with balloons of 5–10 mL capacity (up to 30 mL for fillies) and 12–16 Fr are the best size.
- Catheters made of stiffer material may be necessary for catheterization of neonatal fillies and the placement procedure is technically more difficult. It may be impossible to visualize the urethral opening in a recumbent, neonatal filly. Often the best technique is a blind one, and consists of advancing the catheter tip with the left hand while the fingertips of the right hand (for a right-handed operator) direct the tip ventrally along midline of the vestibular floor. Insertion of a sterile, fine-gauge stylet may greatly facilitate catheter placement in fillies.
- Colt foals are easier to catheterize and the procedure is best performed in lateral recumbency. The end of the penis is grasped and exteriorized from the sheath (this is often the most difficult part of the procedure and a firm grip is required to prevent the penis being retracted). The site is then prepared. The lubricated tip of the catheter is then advanced slowly along the urethra. Resistance will be felt at the pelvic brim and neck of the bladder. Stiffer catheters are again easier to place.
- Sterile saline solution is generally preferred to inflate the balloon once the bladder lumen has been accessed because the weight of the fluid may help the catheter tip to sink, adding stability and decreasing the likelihood that the catheter will be expelled. However, some clinicians advocate the use of air and a larger balloon (30 mL) in filly foals citing the fact that the weight of the saline may predispose to straining and the wider urethra in fillies allows easy passage of a 5–10 mL balloon.
- Proper placement of the catheter can be promptly verified with transabdominal ultrasonographic imaging; the fluid-tipped catheter can be clearly imaged in situ.
- Suturing of the catheter to the preputial or vulvar skin is not generally required but using a bandage to attach the catheter to the tail in fillies may help prevent displacement. It is important to ensure that such a bandage is not placed so tightly as to occlude coccygeal blood flow.

Nasogastric intubation (Fig 3.79)

Passage of a nasogastric tube may be required for a one-off administration of colostrum or for continued feeding in critically ill foals (see Chapter 4, p. 129). A variety of tubes are available and selection of a particular tube is often based on clinician preference. Some considerations in selection and use are as follows:

- Stiffer tubes are often easier to place – placing the tube in cold water prior to placement may increase stiffness.
- The smallest diameter tube possible should be used for long-term placement. This is particularly important as the clinical condition of the foal improves and it commences normal feeding in association with tube feeding.
- The required length should be measured prior to placement and the tube marked at the appropriate length. This is done by measuring from one hand-width behind the triceps along the neck and down

Figure 3.77: Placement of a urinary catheter in a filly foal. See text for description of the technique.

Figure 3.78: Urethra of a male foal through which the urinary catheter is passed.

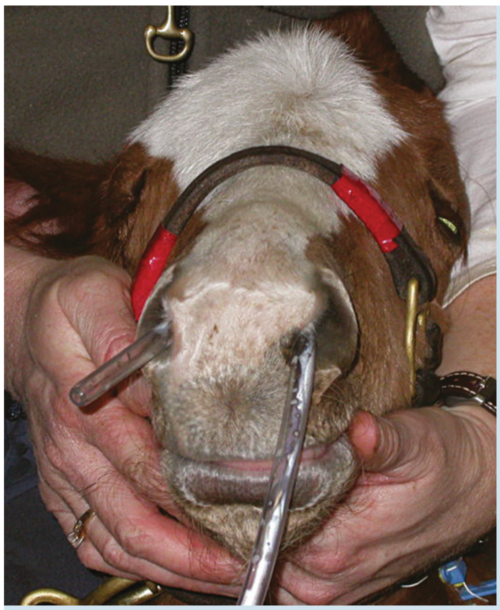


Figure 3.79: Return of a nasogastric tube down the opposite nares from which it was passed – if this occurs the tube should be removed gently.

the head following the path of the esophagus. With minimal practice this can be a very useful technique.

- The ideal site for placement is a controversial issue. Some clinicians prefer the distal esophagus and some prefer to pass through the cardia into the stomach – either location carries the risk of aspiration and the second method may have an increased risk of gastric irritation. The second method is easier to confirm placement. Those that prefer the latter method are also those that advocate confirming the position of the tube prior to each feeding. Even tubes that have been secured to the skin have been known to slip through the adhesive tape over time resulting in improper positioning which if not detected may result in aspiration of feed.
- Tubes are usually attached to the skin of the nostril via a “button” suture and should also be taped to the headcollar or face (see Fig 3.65).
- The tube should be clamped closed when not in use.
- Feeding should be done by gravity flow and only with the foal in a standing or sternal position. ***NEVER*** feed a foal in lateral recumbency.
- Confirming correct placement:
  - On entry into the stomach, bubbling gas sounds can usually be heard.
  - A gastric odor may also emerge from the tube.
  - Fluid may reflux in certain situations, e.g. pyloric stenosis.
  - Blowing on the tube should produce auscultation of bubbling gas sounds over the stomach.
  - If you can blow but cannot suck on the tube then it is most likely in the esophagus.
  - If you can blow and suck on the tube then it is likely to be in the trachea. Unlike adults young foals will not always cough when a tube is placed into the trachea.
  - If you cannot blow or suck on the tube and there is no audible bubbling then it has kinked over on itself and should be removed slowly and carefully.
  - Radiography will also confirm the positioning of the tube, especially if it is a specially designed tube with a radio-opaque tip.

Fluid therapy (Figs 3.80–3.84)

The goal of fluid therapy in animals of all ages is to expand vascular volume in order to restore and then maintain cardiovascular function, thereby improving organ perfusion and blood pressure while correcting dehydration and disturbances of acid–base balance, osmolality and electrolytes.

The initial formulation of a fluid plan should be based on careful clinical examination of the patient and laboratory parameters (Table 3.3).

It is important to bear in mind that many other factors including the disease process in question may affect many of the laboratory parameters:

- PCV can be increased by excitement and decreased by hemorrhage. Changes in PCV should always be considered in conjunction with the TP. Dehydration will increase both the PCV and TP. Excitement will increase the PCV but not the TP. Early stages of hemorrhage will have a slightly decreased PCV and normal TP but as the hemorrhage continues the TP will drop along with a continuing decrease in PCV. Also, in cases of hemorrhage the decrease in PCV and TP is usually greater than can be explained by fluid therapy alone.

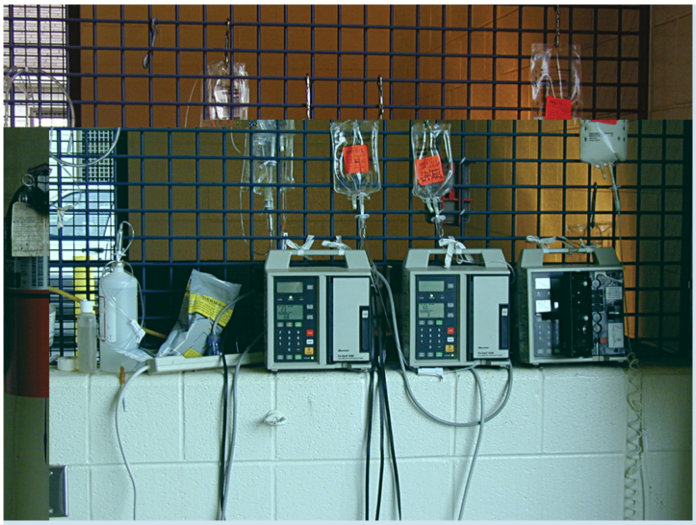


Figure 3.80: Rate control fluid pumps.

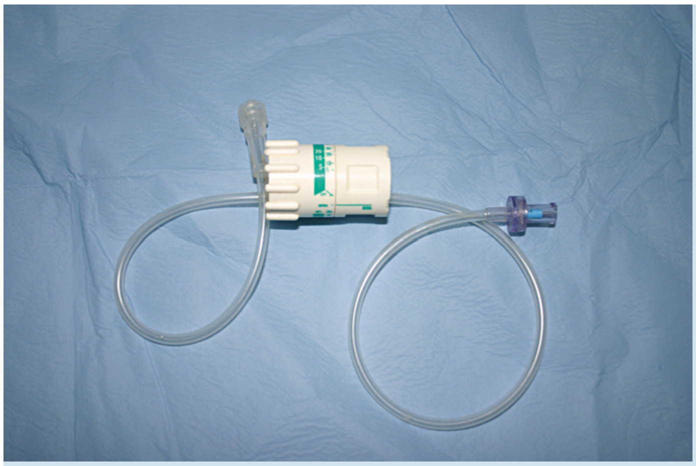


Figure 3.81: MILA rate control fluid dial.

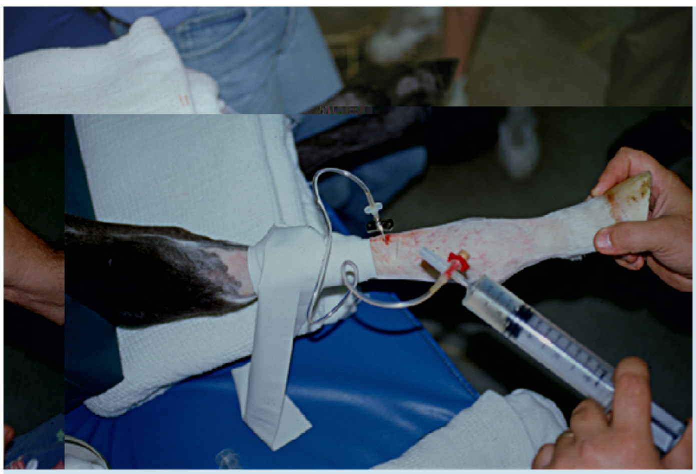
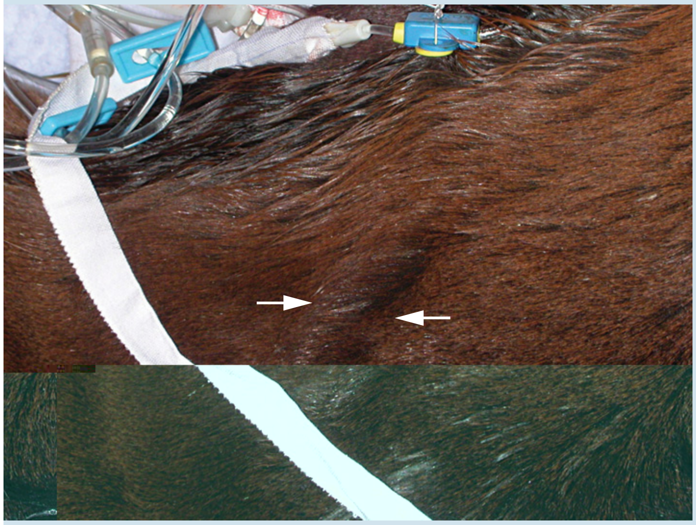


Figure 3.82: Intraosseous fluid administration.



Figures 3.83 & 3.84: Dehydration as evidenced by marked skin tenting (arrows) and a sunken eye (different foals).

Table 3.3: Clinical and laboratory parameters used to assess dehydration

DEHYDRATION (%)	SKIN TENT (SECONDS)	MUCOUS MEMBRANE MOISTURE	CRT (SECONDS)	PCV (%)	TP (g/L)
5	2–3	Moist	1–2	32–40	65–70
10	3–5	Sticky	2–4	36–48	70–75
15	>5	Dry	>4	>48	>75

- TP is also often lower in the normal neonate than in the adult. Failure of passive transfer will also result in lower than normal levels. Severe diarrhea or malnutrition can also result in below normal levels. Increases in TP may be as a result of elevated fibrinogen levels in the neonate or elevated gamma globulins in older foals.
- *Both PCV and TP may be within normal ranges in severely dehydrated animals and should always be considered in conjunction with other clinical and laboratory findings.*
- Azotemia in foals is not always a good indicator of dehydration. Azotemia may be caused by placental insufficiency or renal disease itself. Azotemia caused by placental insufficiency is often (but not always) greater than that which would be expected with dehydration. Pre-renal azotemia will be corrected quickly with appropriate fluid therapy whereas renal azotemia is usually slower to correct.
- Heart rate is not a good indicator of dehydration as it may be influenced by other factors. Handling alone may result in marked elevation of heart rates in normal neonates.
- Urine output and urinary specific gravity (SG) can be very useful means of assessing fluid therapy. They are not so useful in the initial formulation of a fluid plan as frequently the starting point is not known (it is hard to assess increased urine production if the volume of urine being passed initially is unknown) and both measurements can be affected by other factors such as renal disease (see Chapter 6 p. 169).
- Therefore the initial fluid plan is often formulated based on many subjective factors. However, the keyword is "initial"; after fluid therapy has started the foal should be reassessed and the plan restructured if required. Blood work may also be repeated after initial fluids have been administered.

The "rough guide" to fluid therapy

Water deficit (L) = percentage dehydration × body weight (kg)

Thus a 10% dehydrated foal will have a fluid deficit of 5 L.

- Maintenance fluid requirement of a newborn foal is 80–120 mL/kg/day. Therefore a 50 kg foal needs 4–6 L of fluid per day to maintain bodily function. It is important to remember that healthy sucking foals will most likely consume much more than this. A 50 kg foal consuming 15% of its body weight will have a daily fluid intake of 7.5 L.
- Urinary output closely approximates fluid intake and in the healthy foal will be 4–6 mL/kg/h.
- Foals that present without significant reported or detected acid–base or electrolyte abnormalities and mild to moderate dehydration (5–10%) will usually receive an initial 1 L of crystalloid fluid such as lactated Ringer's or normosol, over 1 hour and then be reassessed.
- **Any neonatal foal that is not nursing should receive fluid therapy. A "wait and see" policy will not work!**
- The initial fluid replacement will in many cases improve attitude and appetence. A foal which may not have been nursing on arrival may start to do so, leading to correction of some of its fluid deficit. Rarely will a foal require its entire deficit to be replaced by intravenous fluids. In fact doing so may lead to overhydration which in compromised foals could result in generalized edema.
- Normal saline (0.9%NaCl) is indicated for hyperkalemia, hyponatremia, hypochloremia and metabolic alkalosis. It should not be used as a maintenance fluid.
- Dextrose (5%) is often added to other fluid types and such fluids can be used in cases of hypoglycemia or hyperkalemia. It should be noted that fluids containing 5% glucose will not be able to meet the nutritional requirements of the foal. (See Chapter 13, p. 324).
- Hypertonic saline (7% NaCl) can be used in cases of acute blood loss and shock at a dose of 2–4 mL/kg. However, it must be rapidly followed by either oral administration of fluids or combined oral and rapid intravenous fluid administration. The administration of hypertonic saline should be carefully considered in neonates as many of the contraindications for its use (such as seizures, hypernatremia, hypokalemia, renal failure and thrombocytopenia) are frequently encountered. It should be considered for situations

where rapid stabilization of the cardiovascular system is required and where failure to do so is likely to result in death.

- Colloids (e.g. Hetastarch 10 mg/kg) are substances with a large molecular weight that remain restricted to the plasma compartment after administration. Plasma is the most frequently administered colloid in neonates and has the added benefit of providing immunoglobulins.
- When considering a fluid plan the type of disease process involved and continued losses should be taken into account. The most important example of such in neonates is the foal with severe diarrhea. Commonly these foals cannot tolerate oral administration of fluids and have high fluid losses. The fluid requirements of these foals can be 2–3 times maintenance or more.
- Ideally fluids should be continually administered through flexible tubing that allows for movement of the foal. This may not be suitable in all cases and thus the planned fluid should be administered in 6–8 doses per day.
- When devising a plan it is important to include all sources of fluids in your calculations. Calculate the total that will be administered orally and intravenously and allow for additional fluid sources such as parenteral nutrition.

Administration of bicarbonate

- Severe diarrhea results in metabolic acidosis from a loss of bicarbonate ions and lactic acidosis as a result of poor perfusion. Bicarbonate therapy is indicated when pH < 7.2 or the base deficit is >10 mEq/L.
- Calculation of bicarbonate deficit:

$$\text{body weight (kg)} \times 0.6 \text{ extracellular fluid (ECF)} \times \text{deficit (normal} = 25 \text{ mEq/L)}$$

- Calculation of the base deficit should be part of the daily laboratory assessment of critically ill foals and those with diarrhea. In less than ideal situations where daily measurement is not possible due to the lack of laboratory facilities or a remote location, a significant base deficit should be suspected in foals that are hyperventilating in the absence of respiratory disease. However, bicarbonate administration should be based on the calculated deficit as overzealous administration can lead to metabolic alkalosis.
- Commercially available preparations are hypertonic (5% $NaHCO_3$, 8.4% $NaHCO_3$) and must be diluted prior to administration. To make isotonic $NaHCO_3$ (1.3%) add 260 mL of 5% $NaHCO_3$ or 154 mL of 8.4% $NaHCO_3$ to a liter of sterile water. If adding bicarbonate to other fluids carefully calculate the mEq of sodium that will be in the final mixture as it is easy to induce hypernatremia which may prove difficult to correct.
- TCO_2 may be used to estimate HCO_3. Generally, HCO_3 is 1–2 mEq/L < TCO_2.
- *Do not use bicarbonate in solutions containing calcium because an insoluble calcium carbonate precipitate will form.*
- Administer one-third to one-half the calculated deficit during a 30-minute period, then re-evaluate or administer the remainder slowly during a 12–24-hour period. If respiratory compromise is present administer one-third to one-half during a 50-minute period.
- Monitor blood gases if you administer >1–2 mEq/kg over a short period.

Intraosseous fluid administration

- Intraosseous administration of fluids and antibiotics can be used in foals in which venous access is not available.
- Intraosseous needles are available but in an emergency a 14G 1.5″ needle can be used in the medial aspect of the proximal tibia.
- The mid cortex of any long bone can be used. The tibia or radius are the most common sites but a metacarpal or metatarsal bone can also be used.
- The site should be clipped and surgically prepared. Local anesthetic is then injected at the site following by a stab incision through the skin and periosteum.
- A slow speed drill is then used to create a drill hole into the medullary cavity. The drill hole is then tapped with a suitably sized instrument.
- The infusion screw is then placed in the hole and tightened so that the head is outside skin level.
- A dressing is then applied to protect the whole region but the infusion port should remain accessible.
- A mild swelling should be expected at the site for 1–2 months following the removal of the screw.

Nursing care of critically ill foals (Figs 3.85–3.89)

Bedding

- Critical foals should be placed on a soft mattress with a waterproof covering to facilitate cleaning. Warm, dry blankets should be used over and under the foal. Pillows and foam padding can be used to help maintain a sternal position. Such foals are often separated from their dams by stall dividers although many mares will tolerate a mattress in their stall.
- The entire bedding should be changed every 24 hours and protective absorbent bedding should be changed whenever soiled.

Position

- Sternal recumbency allows best ventilation but the direction in which the foal's hind legs are facing should be changed at least every 2 hours and ideally every hour. If possible the foal should be encouraged to stand, supported where necessary, for 5 minutes every hour.
- Physiotherapy including passive range of motion exercises and massage of large muscle masses may help to stimulate circulation.
- Joints should be checked daily for any heat or swelling.

Umbilicus

- The umbilical stump should be treated with 0.5% chlorhexidine solution 4–6 times during the first 24 hours of life; 2–3 times daily thereafter for 2–3 days should be adequate in healthy foals. Hospitalized foals may require continued treatments. To improve desiccation of the umbilical stump adding surgical spirit to the solution is now recommended. The required solution can be made by mixing 125 mL of 2% chlorhexidine solution with 300 mL of sterile water and 75 mL of surgical spirit.
- The umbilical stump should be inspected daily for signs of swelling, infection or patent urachus. If in doubt ultrasound it.

Eyes

- The eyes should be inspected 4–6 times daily for evidence of trauma, corneal abrasions or entropion.

Figure 3.85: Use of a foam "V" as an aid to maintaining sternal recumbency in a foal. Also in this image note the nasogastric and intranasal oxygen tubes, the presence of a urinary collection system which is fastened to the thigh to help prevent displacement, the presence of an abdominal bandage following colic surgery, and the incontinence pads which are used to help prevent soiling of the bed and as an aid to keeping the foal dry.

Figure 3.86: Use of a special form-fitting foal bed to maintain sternal recumbency.

- Antibiotic ointment or artificial tears should be applied at each examination in recumbent foals.

Perineum and sheath

- If diarrhea is present the perineum should be cleaned, thoroughly dried and a petroleum-based cream applied 4–6 times daily to avoid scalding.
- Urinary catheters should only be used when medically necessary and should be inspected frequently for correct placement and the absence of obstructions.

Intravenous catheters

- The catheter site should be inspected frequently and if a dressing is present over the catheter it should be changed daily.

Figure 3.87: Use of blankets to maintain body temperature.

Figure 3.88: Correct positioning for feeding a foal with a bottle.

Figure 3.89: Checking for the presence of reflux prior to feeding a recumbent foal. Note the measuring cup. All reflux should be measured to assess whether it is increasing or decreasing over time.

- Catheters should be changed every 72 hours unless specifically designed for longer placement.

Fluid administration sets

- Change fluid administration sets daily if glucose containing fluids are being administered. If the line remains intact every 48–72 hours is sufficient.
- The use of a harness and coiled giving sets facilitate fluid administration in ambulatory foals.

Oxygen administration equipment

- Change oxygen insufflation lines and humidifiers every 24–48 hours.
- Nasal insufflation lines should be inspected 4× daily to ensure that holes have not become occluded by mucus.
- The lines should be checked and drained of any built up condensation frequently.

Feeding the sick foal

See Chapter 4, p. 129.

Recommended reading

Acworth NRJ 2003 The healthy neonatal foal: routine examinations and preventative medicine. Equine Veterinary Education 15(4):207–211

Corley KT, Furr MO 2000 Cardiopulmonary resuscitation in newborn foals. Compendium on Continuing Education for the Practicing Veterinarian 22:957–966

Langdon Fielding C, Magdesian KG 2003 Cardiopulmonary cerebral resuscitation in neonatal foals. Clinical Techniques in Equine Practice 2(1):9–19

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Seahorn TL, Cornick-Seahorn J 1994 Fluid therapy. Veterinary Clinics of North America: Equine Practice 10(3):517–525

Sprayberry KA, Carr EA 2003 Evaluation and early care of the sick neonatal foal. Current Therapy in Equine Medicine 5:631–635

Wilkins PA 2004 Disorders of foals. In: Reed SM et al (eds) Equine internal medicine. Saunders, St Louis, p 1381–1431

CHAPTER 4

The gastrointestinal system

J Scott Weese DVM DVSc DACVIM

Introduction

Disorders of the gastrointestinal system are extremely common in foals of all ages. A wide variety of disorders can be encountered with a broad range of etiologies. A number of diagnostic tests and procedures are available that not only help the clinician reach a correct diagnosis but may also help in monitoring disease progression.

Diagnostic tests

Abdominocentesis and abdominal fluid types (Fig 4.1)

Abdominocentesis can provide useful information in foals with gastrointestinal disease, particularly differentiating abdominal disease from uroperitoneum and aiding in the decision whether or not to perform an exploratory celiotomy. Ideally, abdominocentesis should be performed following ultrasonographic examination of the abdomen to identify pockets of peritoneal fluid.

Peritoneal fluid cannot always be obtained, regardless of the disease process, and the ability or inability to collect fluid has no prognostic value.

Method

1. The usual site is midway between the umbilicus and the xiphoid, to the right of midline to avoid inadvertent penetration of the spleen.
2. Aseptic preparation is extremely important, clip and scrub the site.
3. Identify a fluid pocket via ultrasonography.
4. A 21G, 5–7 cm needle or a bovine teat cannula can be used. A teat cannula carries less risk of traumatizing the bowel but a stab incision through the skin and muscle is required first.
5. If fluid does not flow freely carefully aspirate with a 5 mL syringe.
6. Collect fluid into both an EDTA and plain tube.

Interpretation

- Visual examination, determination of total protein and white cell count can provide most of the relevant information. Normal

Figure 4.1: Abdominocentesis being performed in the left ventrolateral abdomen. This site was chosen in this foal on the basis of ultrasound findings of a localized peritonitis.

peritoneal fluid is clear to yellowish with a total protein of less than 25 g/L and less than 5×10^9 WBC/L.
- With intestinal compromise, fluid color will change from clear to dark yellow, orange or reddish. Turbidity, total protein and white blood cell count will increase.
- With enteritis, mild to moderate increases in total protein with lesser visual changes may occur.
- If peritonitis is present, fluid will be orange-red to milky, very turbid and possibly foul-smelling. Ingesta may be observed in foals eating a solid diet in which rupture of an abdominal viscus has occurred. It is critical that the clinical condition be taken in consideration when evaluating the results. Enterocentesis can occur, and should especially be considered in cases where the severity of disease is not consistent with severe septic peritonitis.
- WCC >10,000 cells/μL, protein >3 g/dL indicate peritonitis. Serum to peritoneal fluid glucose differences of >50 mg/dL are suggestive of peritonitis. Peritoneal fluid glucose <30 mg/dL, pH <7.3 and fibrinogen concentration >200 mg/dL are indicative of peritonitis.
- Identification of intracellular bacteria and degenerate neutrophils on cytological analysis of peritoneal fluid is consistent with peritonitis.
- Iatrogenic blood contamination from body wall vessels or splenic puncture can result in abnormal red coloration of normal peritoneal fluid.
- Often, the color of fluid will change during collection. Reddish samples can be centrifuged to differentiate iatrogenic contamination or acute hemorrhage from compromised intestinal viscera.

Ultrasonography

Ultrasound is non-invasive, well tolerated by the foal and easily performed in the field. The small size of the foal abdomen and proximity of many organs of interest makes the foal an ideal candidate for abdominal ultrasonography.

Sector and linear transducers can be used and both produce high quality images. Linear transducers produce a rectangular image that is well suited for examining superficial structures; however, with increasing depth the image of the far field is narrowed which restricts visualization of deep structures. Curved linear transducers provide wide near and far fields of view and the footprint size is usually intermediate.

Sedation is not generally required but may be necessary in certain fractious foals. Optimal image quality is obtained by clipping the hair over the area of interest or the entire ventral abdomen if required. The abdomen should then be washed and ultrasound coupling gel applied to the skin.

Imaging of the abdominal contents

Stomach

The stomach is located medial to the spleen in the mid to ventral abdomen between the 6th and 12th intercostal space (ICS) on the left side. The ventral wall of the stomach is in contact with the ventral abdomen up to 7 days of age. The stomach wall is hypoechoic relative to surrounding structures. In foals less than 7 days old, luminal contents are visible. In older foals the presence of gas prevents imaging of the gastric contents and at this stage the foal's stomach resembles that of an adult with a large curvilinear echo medial to the spleen and caudal to the liver.

Small intestine

The small intestine can be visualized in a larger area of the abdomen in young foals compared with adults. The small intestine has a hypoechoic wall and its lumen is easily observed. The duodenum is visualized between the ventral and caudal aspect of the liver and the dorsal margin of the right dorsal colon (see Figs 4.55–4.57). It can also be seen ventral to the caudal pole of the right kidney and dorsal to the cecal base (see Fig 4.60).

Large intestine

The large intestine is recognized by its larger diameter and sacculated appearance. The lumen of the large intestine is not normally visualized due to the presence of gas.

Spleen

The spleen is imaged between the 7th ICS and the paralumbar fossa (PLF) on the left side and in the 9th ventral ICS on the right side in contact with the liver. The splenic vein is imaged on the medial aspect of the spleen, caudal and dorsal to the stomach in the 11th to 12th mid-ICS (see Fig 4.59). The spleen interfaces caudally with the left kidney.

Liver

The liver can normally be seen in the cranioventral and midabdomen between the 7th and 14th ICS and dorsally in the 14th ICS on the right side of the abdomen. On the left side it can be seen between the 6th and 10th ventrolateral ICS in a larger area compared to mature animals.

Radiography (Fig 4.2)

Radiographs are most easily taken with the foal in a standing position. Clinician preference may largely dictate whether radiography or ultrasonography is used. Radiography is most useful in contrast studies which can be used to diagnose conditions of the esophagus and duodenum, in addition to the use of barium enemas to diagnose conditions of the distal gastrointestinal tract.

Figure 4.2: Positioning for taking abdominal radiographs.

Figure 4.3: Oral examination revealing a cleft hard and soft palate.

Barium swallow

- Give 120 mL barium sulfate suspension orally.
- Take radiographs immediately.

Contrast studies of the stomach / small intestine

- Withhold food for 4–12 hours depending on the age of the foal. Younger foals should be withheld for shorter periods.
- Administer 5 mL/kg of a barium sulphate solution via nasogastric tube.
- Take radiographs at 1, 15 and 30 minutes and then every 2 hours until the barium has passed the area of interest.
- Barium should have left the stomach within $1–1^{1}/_{2}$ hours in a normal foal.

Barium enema

- Administer 180 mL barium sulphate solution via an enema tube.
- Ventrodorsal and lateral radiographs should be taken immediately.

Inherited / congenital conditions

Congenital disorders are present at birth and may or may not be inherited. Inherited disorders may be present at birth (inherited and congenital), e.g. atresia coli, or may manifest later in life, e.g. Culicoides hypersensitivity. Congenital and inherited disorders of the gastrointestinal system are the most frequently encountered of any body system.

Cleft palate (Figs 4.3–4.6)

History

Cleft palate (palatoschisis) is an uncommon problem caused by failure of the closure of the palate during development. The most common type involves the caudal aspect of the soft palate; however the hard palate, lips and external nares may also be affected. The cause is not understood, although genetic and environmental factors may play a role.

Clinical signs

- Bilateral milky nasal discharge is usually observed shortly after nursing. Foals may cough during or after nursing, and milk may be expelled while coughing.
- Aspiration pneumonia is common, and may be associated with cough, fever, increased respiratory rate, and sepsis. Lack of cough does not rule out respiratory disease in foals.
- Cleft palate is not always obvious and may only be suspected in some cases in foals with recurrent or non-responsive pneumonia.
- Dysphagic foals are at high risk for sepsis because passive transfer of maternal antibodies may be affected.

Differential diagnosis

Nasal discharge after nursing and dysphagia may also occur with:

- neurologic dysphagia
- esophageal obstruction
- primary infectious pneumonia
- congenital laryngeal/pharyngeal abnormalities
- severe weakness and depression.

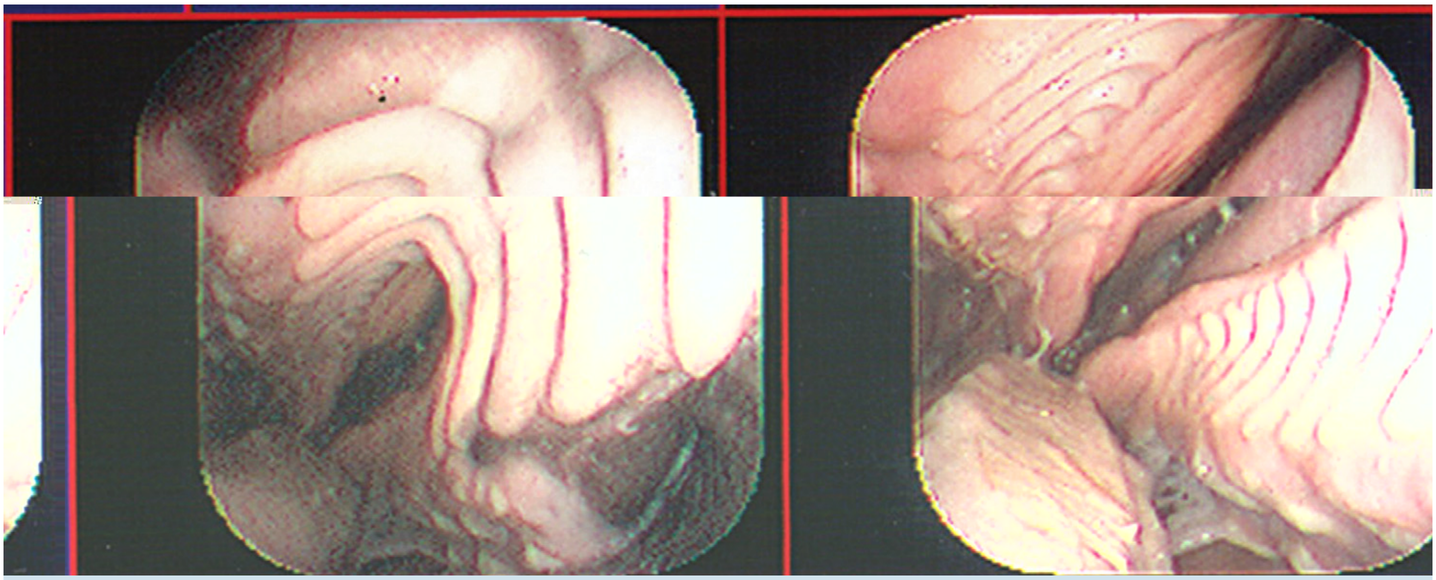
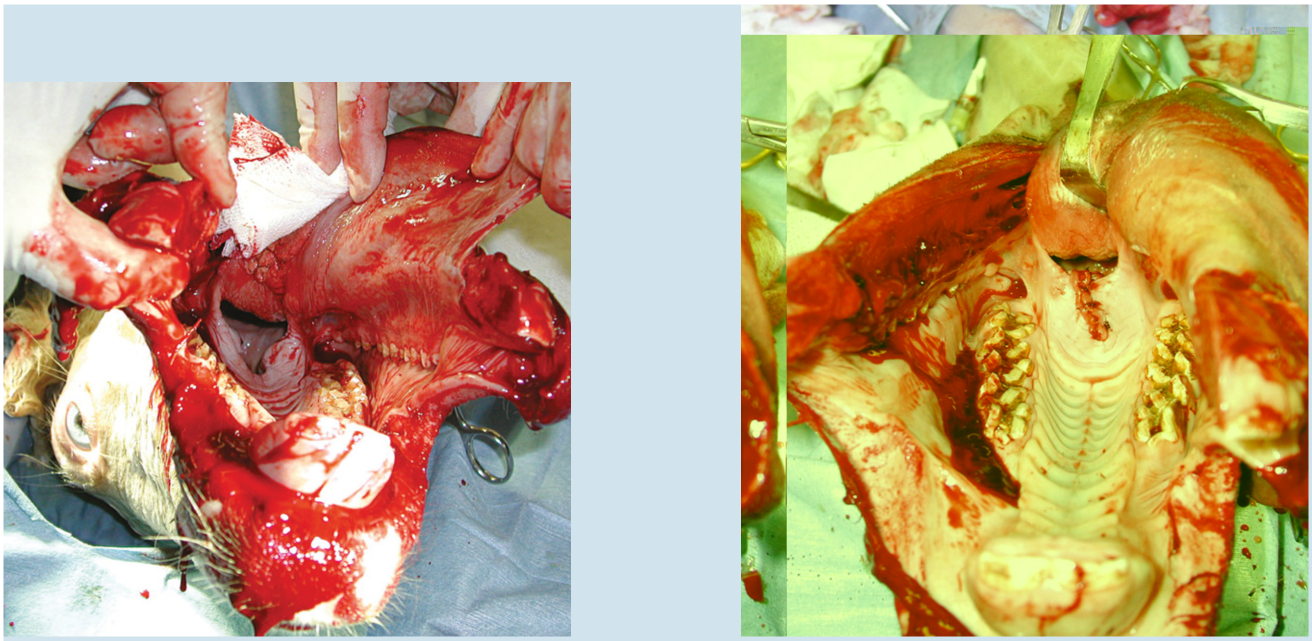


Figure 4.4: Oral endoscopic examination revealing a cleft hard and soft palate.



Figures 4.5 & 4.6: A cleft soft palate prior to and following surgery. As can be seen from these images surgical access is difficult.

Diagnosis

- Thorough neurological examination with particular attention to cranial nerves should be performed to rule out neurological causes of dysphagia.
- In some cases, diagnosis can be made with a careful oral examination.
- Endoscopy should be performed via both nares. Oral endoscopy may also be required.
- Thoracic radiographs are useful to identify and characterize secondary pneumonia.
- Immunoglobulin G levels should be assessed to identify failure of passive transfer of maternal antibodies.

Treatment

- Treatment needs to be directed at two main areas – correction of the abnormality and treatment of secondary problems such as pneumonia, sepsis and malnutrition.
- Treatment of the defect involves one of a variety of surgical techniques. Dehiscence of the surgical site is a common problem. In severely compromised foals, surgery may need to be delayed while

Figures 4.7 & 4.8: Parrot mouth visualized with the lips closed and lips opened. Note the marked disparity in length between the mandible and maxilla.

secondary problems (pneumonia, sepsis) are addressed. In these situations, feeding via a nasogastric tube or parenteral nutrition is required.

- Some degree of pneumonia will almost invariably be present. Broad-spectrum antimicrobial therapy is indicated. Culture and sensitivity testing of a tracheobronchial aspirate is useful. Prolonged treatment may be required in foals with severe pneumonia or lung abscesses.
- A plasma transfusion is indicated if failure of passive transfer of maternal antibodies is present. Aggressive supportive care may be required in some cases.
- The prognosis is better in foals with defects only involving the soft palate.

Parrot mouth (brachygnathia) (Figs 4.7 & 4.8)

History

Parrot mouth (brachygnathia) is a common congenital abnormality characterized by disparity of the lengths of the mandible and maxilla. The mandible is shorter than the maxilla and there is no occlusal contact between the upper and lower incisors. The abnormality may be present at birth or develop over time. Males are more commonly affected.

Clinical signs

In many, if not most cases, no clinical abnormalities will ever be evident. In some cases, problems will not be encountered until the foal begins to eat solid food. With severe defects, nursing and prehension may be impaired. Dental abnormalities from malocclusion may cause problems over time.

Diagnosis

Oral examination is diagnostic and there are no differentials. Malocclusion of the incisors occurs because of misalignment of the upper and lower jaws.

Treatment

- In many cases, parrot mouth is more of a cosmetic defect than a true medical problem. The treatment approach is dependent on the severity of the lesion and the age. Use of a bite plate has been described to help reduce progression of the lesion in skeletally immature horses.
- Surgical intervention may be required in some cases. One approach involves application of a premaxillary tension band to restrict rostral development of the maxilla as the mandible grows normally. This may be useful for moderate defects and should be performed in foals less than 6 months of age. Surgical lengthening of the mandible is also possible.
- In severe cases, the malocclusion may inhibit nursing and/or prehension, particularly grazing, and the diet may need to be modified to facilitate ingestion. Young foals may require feeding from a bucket.
- More frequent dental care is required as the horse ages because of the malocclusion of incisors. The heritability of this condition is unknown.

Sow mouth (prognathia) (Fig 4.9)

History

Sow mouth (prognathia) is a congenital abnormality characterized by malocclusion of the premaxillary incisors because of undergrowth of the maxilla, or overgrowth of the mandible. It is usually identified at, or around, the time of birth but is encountered much less frequently than brachygnathia.

Figure 4.9: Sow mouth in a 1-day-old foal.

Figure 4.10: Dentigerous cyst with obvious draining tract.

Figure 4.11: Radiograph of a dentigerous cyst with a tooth-like structure.

Clinical signs

Clinical abnormalities are not often present and sow mouth may be strictly a cosmetic defect. In some cases, nursing and prehension may be impaired.

Diagnosis

Oral examination is diagnostic and again there are no differentials. Malocclusion of the incisors because of misalignment of the mandible and maxilla is obvious.

Treatment

Treatment is not required unless nursing or prehension is impaired. Bucket feeding of severely affected young foals may be required. Older horses may require a special diet if there are problems with prehension. Frequent dental care may be required. Affected horses may have difficulty grazing short pastures.

Surgical correction is not often indicated. Application of a mandibular tension band can be used to restrict mandibular growth while the maxilla develops normally. Mandibular reduction is very invasive and rarely indicated. A hereditary basis has not been confirmed.

Dentigerous cyst (Figs 4.10 & 4.11)

History

Dentigerous cysts, also referred to as ear teeth, aural fistulae or heterotropic polyodontia, are congenital defects characterized by an epithelium-lined cavity containing embryonic teeth. They are most commonly located adjacent to the temporal bone, but can be found in a variety of other areas of the head. Cysts contain a seromucoid fluid and often fistulate. Dentigerous cysts may be recognized at any age, but are most commonly identified in horses less than 3 years of age.

Clinical signs

A firm, non-painful swelling is typically present at the base of the ear. The mass usually enlarges over time. Bilateral cysts are uncommon but have been encountered. Other clinical abnormalities are uncommon. Serous fluid may be discharged from a draining tract.

Differential diagnosis

Other common causes of a soft tissue mass in this region include:

- abscess
- seroma
- hematoma
- foreign body
- sequestrum.

Diagnosis

- Aspiration of the cyst will produce a pale yellow, serous fluid. No growth is obtained on bacterial culture unless a draining tract is present.
- Ectopic dental tissue (radiodense masses) may be evident on radiographs of the area.
- A contrast fistulogram can be used to delineate the mass and any draining tracts.
- Ultrasonography can be used to characterize the contents of the mass.

Treatment

Surgical excision is indicated. Care must be taken because of the significant blood vessels and nerves in the affected area. Some ectopic teeth can be firmly attached and difficult to remove. Complete removal of the ectopic tissue is required. Without complete excision, recurrent draining tracts are likely.

Prognosis following removal is very good and there is no evidence that this is a heritable condition.

Intestinal atresia – atresia ani / atresia coli (Figs 4.12–4.21)

History

Both disorders are uncommon congenital abnormalities. Atresia coli, although rare, is the most common type of intestinal atresia and may be related to vascular accidents during intestinal development. Affected foals may appear to be normal for the first 24 hours of life.

Figure 4.12: Atresia ani in a colt foal with an apparent absence of an anal opening.

Figure 4.13: Same foal as Fig 4.12 following surgical creation of an anal opening. An incision was made through the skin and underlying mucosa. The mucosa was then sutured to the skin.

Figure 4.14: Atresia ani in a filly foal. This filly had an external anus that was not patent. At the time the photograph was taken the anus had already been made patent by an incision. The filly prior to presentation had developed a rectovaginal fistula allowing feces to be passed through the vagina.

Clinical signs

- After a normal birth and perinatal period, affected foals begin to deteriorate by approximately 24 hours of age. As feces and gas accumulate proximal to the blind-ended anus or section of colon, progressive signs of abdominal pain and distension develop. Foals may be mildly colicky at the start, with progression to intractable pain over a period of hours to a day or more. Affected foals nurse well originally, but become anorexic as pain progresses. Atresia coli is associated with more severe and earlier onset signs than atresia ani.
- In some fillies with atresia ani, development of rectovaginal fistula will be associated with some relief of pain, depending on the amount of feces and gas that can be passed.

Figure 4.15: Same filly as Fig 4.14. This image clearly demonstrates the rectovaginal fistula (finger) and also shows the presence of an abnormal band of tissue at the vaginal opening.

- In cases of atresia coli in which intestinal rupture occurs, there may be a transient period of improvement, based on lessening of intra-intestinal pressure, followed by rapid deterioration within hours as septic peritonitis ensues.

Diagnosis

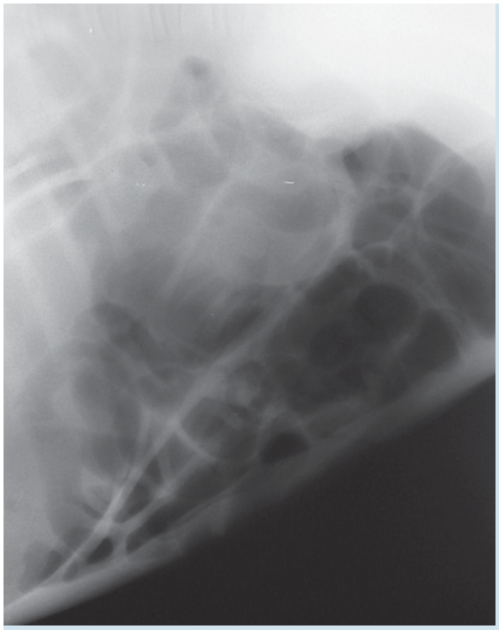
Atresia ani

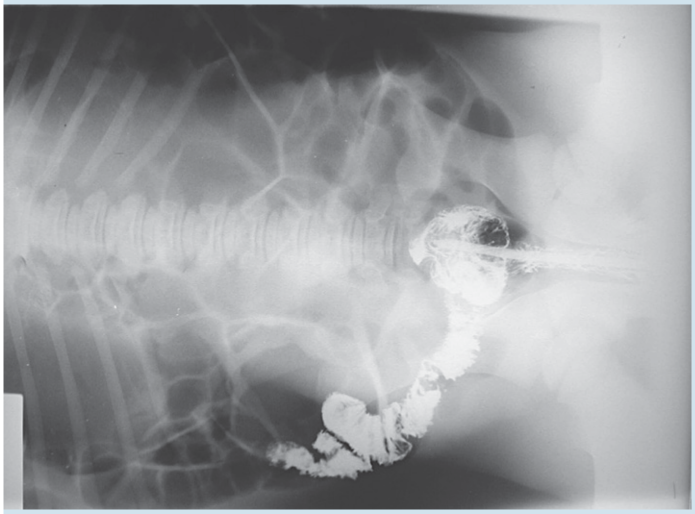
- The perineum should be examined for evidence of a normal anus. In some cases, the anus is absent. In others, an external anus is visible but not patent. If feces are present in or around the vulva, vaginal examination for a rectovaginal fistula should be performed.
- If an external anus is present, a blind-ended structure may be palpable within the rectum.
- Proctoscopy can be used to confirm that a palpable obstruction is indeed atresia ani.
- Foals should be examined for other congenital abnormalities. Rarely, deformities (or absence) of the tail or vagina may also be present.

Atresia coli

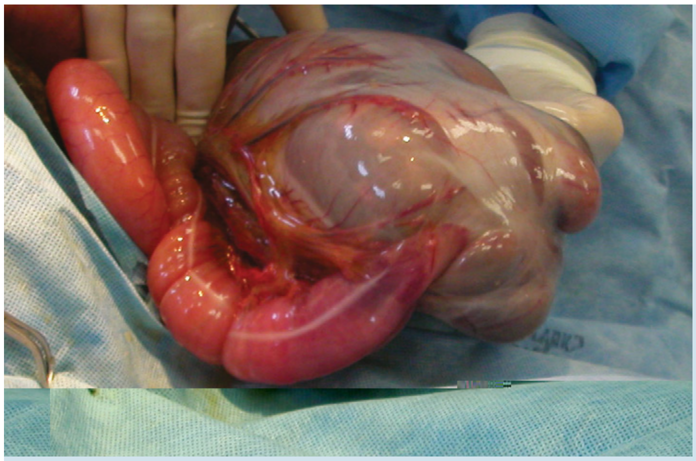
- Diagnosis is more difficult because the lesion is not evident grossly; meconium impaction is often suspected initially because of the failure to pass feces and progressive abdominal pain and distension.
- Digital palpation per rectum: a normal rectum but no feces are present. Note foals with meconium impaction in which no feces are present will still have a small amount of feces colored mucus/fluid and will result in staining of the glove. Foals with atresia coli will only have mucus present and there will be no fecal staining of the glove.
- A blind-ended lesion may be evident with proctoscopy/colonoscopy.
- Distension of the proximal intestine may be evident radiographically.
- Contrast radiographs (barium enema, barium series) can confirm an obstruction but not necessarily atresia coli.

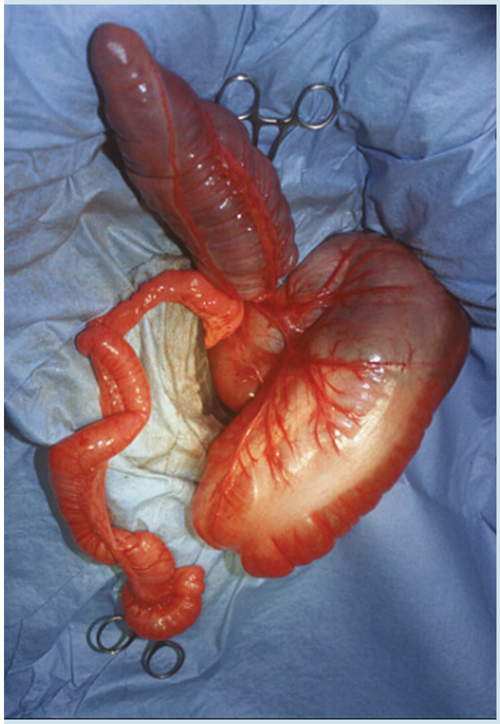
Figures 4.16 & 4.17: A 1-day-old colt foal with atresia coli showing marked signs of colic with repeated efforts to defecate.





Figures 4.18 & 4.19: Radiograph from the foal in Fig 4.16 showing gaseous distension of the colon. The blind ending colon cannot be visualized on plain radiographs, and barium studies (Fig 4.19) are required to confirm an obstruction.





Figures 4.20 & 4.21: At surgery there appears to be an attachment between the large and small colons despite a disparity in size. Dissection reveals that the connection is only a fibrous one and that both structures are blind-ended.

- Exploratory laparotomy may be required for definitive diagnosis.
- Thorough examination for other congenital problems that may accompany atresia coli (i.e. renal aplasia or hypoplasia, hydrocephalus, cerebellar dysplasia) should be performed.

Differential diagnosis

Other causes of progressive abdominal distension without passage of feces in a neonatal foal include:

- meconium impaction
- lethal white syndrome.

If it is unclear whether feces have been passed, other conditions should also be considered, including:

- intestinal accident
- impending enteritis.

Treatment

Atresia ani

- Prompt surgical correction is required, and has been successful in some cases.
- The prognosis with surgery is dependent, in part, on the amount of intestine involved and whether concurrent abnormalities are present.
- The prognosis is better when the anal sphincter is normal and the obstruction is only a thin, bulging layer of tissue.
- Affected horses should not be bred because of the possibility of a genetic basis.

Atresia coli

This condition should be regarded as fatal. Intestinal motility problems or failure of the anastomosis have resulted in poor outcomes in cases in which surgical correction has been attempted.

Lethal white syndrome (Figs 4.22–4.24)

History

Also known as ileocolonic aganglionosis, lethal white syndrome is an inherited, congenital condition that occurs in foals that are homozygous for the lethal white gene. It is an autosomal recessive condition that means that if two carriers (heterozygotes) are bred approximately 1 in 4 of their offspring will be homozygous or affected. Heterozygotes usually have the Overo colour pattern, which is most common in American Paint horses but may also be found in Quarter horses, Pintos and Saddlebreds. White foals born to other breeds (e.g. thoroughbreds) are not affected.

Clinical signs

Most affected foals are entirely white with white irises, but some may have small areas of pigmentation on the forelock and tail. Within 4–24 hours of life, abdominal distension and pain develop. These signs are progressive and become very severe. Minimal feces are passed.

Differential diagnosis

Other causes of progressive abdominal pain and minimal passage of feces include:

- intestinal accident
- intestinal atresia
- meconium impaction
- impending enteritis
- peritonitis.

Figure 4.22: Overo mare with lethal white foal.

Diagnosis

- A white foal born to an Overo–Overo mating with progressive abdominal distension and minimal fecal production is highly suggestive. This history and progression of clinical signs is typically used to make a diagnosis.
- Diagnostic imaging (ultrasound, radiography) demonstrate gaseous intestinal distension.
- Genetic testing can determine whether the foal is homozygous for the condition but is impractical considering the time delay.

Treatment

None. This is an invariably fatal condition and affected foals should be promptly euthanized.

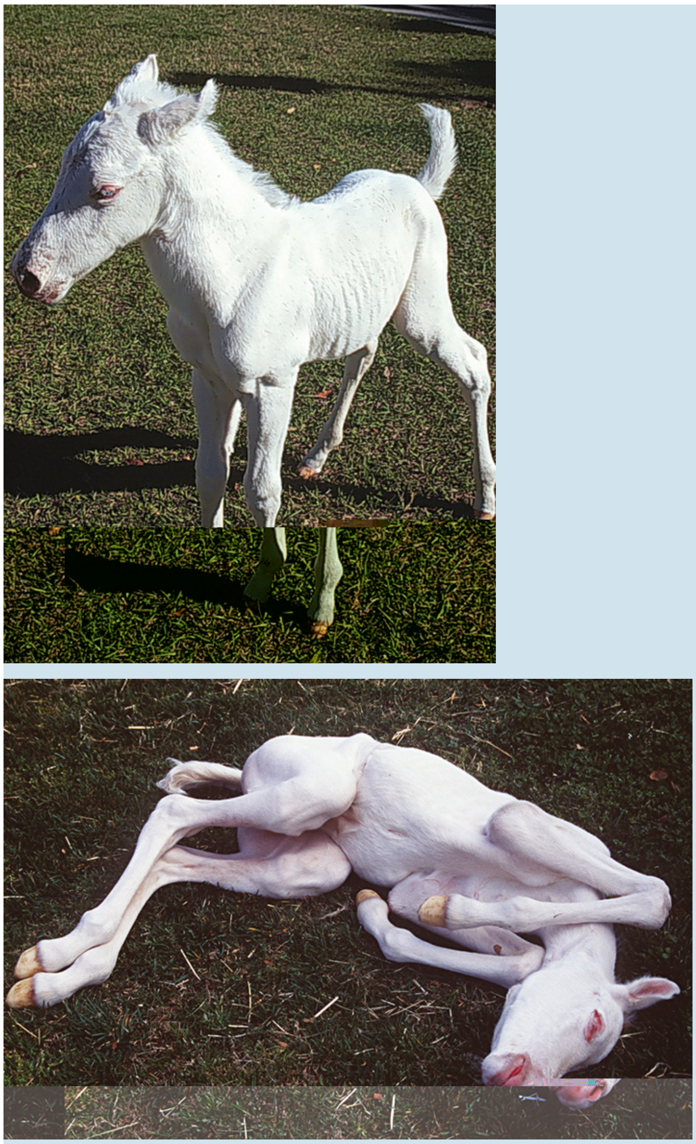


Figure 4.23: Lethal white foals showing typical colic signs.

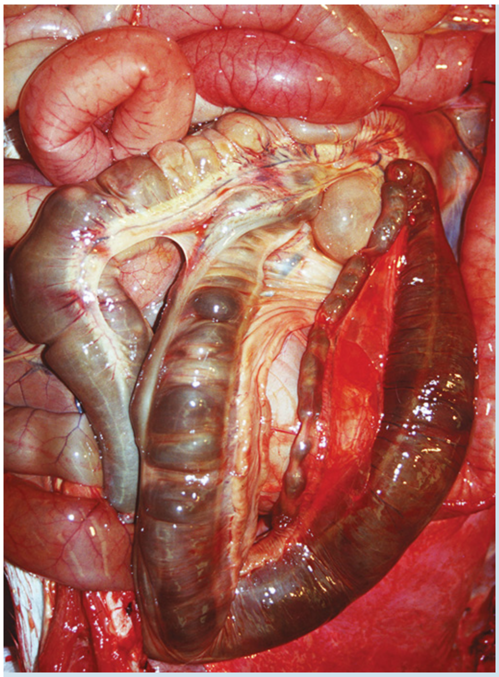


Figure 4.24: Hypoplastic colon with marked discoloration in a lethal white foal.

Figure 4.25: Marked tenesmus in a foal with a meconium impaction.

Figure 4.26: Continued straining associated with meconium impaction may result in rectal prolapse.

Meconium impaction (Figs 4.25–4.29)

History

Normally, meconium is passed within a few hours of birth. Incomplete passage of meconium can result in abdominal pain and distension in foals ranging from <1 day to 2 days of age. Meconium may be impacted anywhere from the large colon to the rectum. Colts are more commonly affected as they have a narrower pelvis than fillies.

Clinical signs

Signs of abdominal pain, including dorsal recumbency, rolling, flank watching, anorexia, tail swishing, tail raising, and tenesmus are common. Foals should be clinically stable otherwise, with no evidence of cardiovascular compromise or toxemia.

Differential diagnosis

Other causes of abdominal pain in neonatal foals include:
- impending enteritis
- intestinal accident, particularly small intestinal volvulus
- ileus.

Abdominal distension and lack of feces can also occur with:
- atresia ani
- atresia coli
- lethal white syndrome.

Diagnosis

- Signalment and a history of minimal or no passage of meconium is often used for a subjective diagnosis. A history of passing a small volume of meconium does not rule out meconium impaction.
- Meconium is sometimes palpable on digital rectal examination.
- Meconium impaction can often be visualized with abdominal radiographs.
- Barium enema can be useful to identify impactions that are not convincing on survey radiographs.
- Abdominal ultrasonography is useful to rule out other causes of colitis, but may not consistently confirm an impaction.
- A nasogastric tube should be passed to determine whether gastric reflux is present. Nasogastric reflux is uncommon with meconium impaction and is usually more indicative of a more proximal obstruction, e.g. small intestinal intussception.

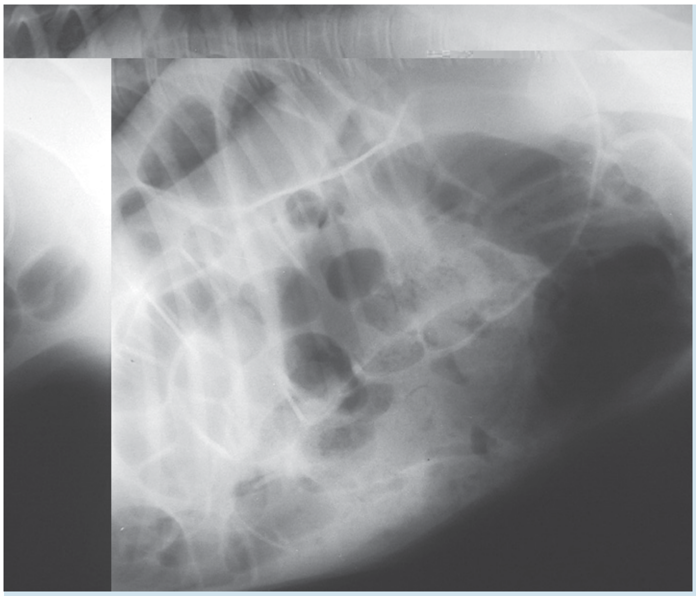


Figure 4.27: Abdominal radiograph from a foal with a meconium impaction demonstrating gaseous distension of the colon.

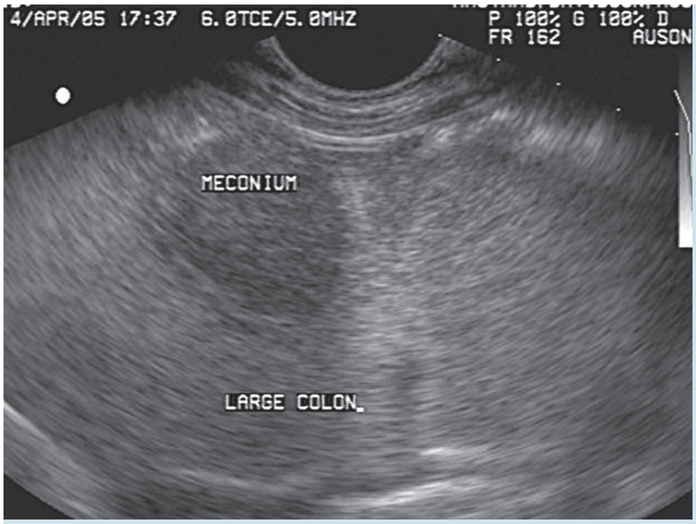


Figure 4.29: Ultrasonographic image of the abdomen demonstrating the presence of a ball of meconium within the colon surrounded by feces of a more fluid consistency.

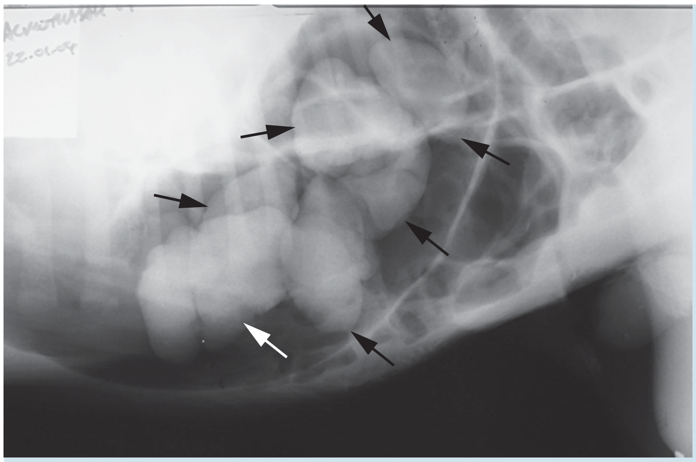


Figure 4.28: Abdominal radiograph from a foal with a meconium impaction demonstrating gaseous distension of the colon and the presence of meconium (arrows).

Figure 4.30: Administration of a commercial enema.

Treatment (Figs 4.30–4.32)

- In mild cases, a single enema (mild soap and water, light lube and water, commercial enema) may resolve the impaction. Concurrent administration of *N*-butylscopalammonium bromide (Buscopan®) (20 mg/dl) at a dose of 0.5- 0.75 cc per 50 kg IV may decrease colonic spasm and aid in passage of the meconium. Low-level exercise (paddock turnout) may also aid passage of the meconium following the administration of an enema.
- Irritating solutions should not be used and repeated administration of commercial enemas should be avoided because of possible phosphate toxicity.
- If there is poor response to a routine enema, an acetylcysteine enema (250 mL 4% acetylcysteine) is indicated. *N*-butylscopalammonium bromide is used by the editors to decrease colonic spasm and thereby allow the enema to work more effectively. The solution is administered via gravity flow through a Foley catheter. The catheter is then clamped and left in place for 10–15 minutes after which time it is removed. Sedation with butorphanol and/or torbugesic (see Appendix 5) is also useful as it not only facilitates passage of the enema but also provides analgesia. If the initial acetylcysteine is unsuccessful a second treatment can be attempted after 1 hour.
- Concurrently, water or mineral oil can be given via nasogastric tube.
- If nursing is restricted, fluid and nutritional support may be required.

Figure 4.31: Administration of a soapy enema using a flexible tube inserted into the anus.

Figure 4.32: Administration of an acetylcysteine enema. This foal has been sedated and placed over the bale as a means of elevating the hind quarters.

- Conservative over-hydration with intravenous fluids may also be useful in cases that are refractory to initial treatment.
- If there is no gastric reflux and a surgical lesion is not suspected, allowing the foal to nurse may be useful to help stimulate intestinal motility.
- Occasionally, surgical intervention is required because of severity of pain or poor response to medical treatment. At surgery, saline can be injected into the affected area and the bowel massaged to relieve the impaction without need of an enterotomy.

Acquired conditions of the gastrointestinal system

Megaesophagus

History

Megaesophagus is a rare condition characterized by chronic dilation of the esophagus. There are a variety of possible causes – esophageal obstruction, chronic gastroesophageal reflux and severe gastric ulceration. Severe gastric ulceration with concurrent gastroesophageal reflux is likely the most common cause. Primary congenital motility disturbances, either neurogenic or myogenic in origin, are extremely rare. Foals of any age can be affected.

Clinical signs

Dysphagia, regurgitation, nasal discharge (often containing feed material), depression, weakness, and hypersalivation are common. Aspiration pneumonia may be present, accompanied by fever, cough, and increases in respiratory rate and effort. Colic may be present with gastric ulceration. In chronic cases, affected animals may have weight loss, ill-thrift, and poor growth.

Differential diagnosis

Dysphagia and nasal discharge can also be present with:

- cleft palate
- esophageal obstruction
- pharyngeal dysfunction
- severe pneumonia with dysphagia caused by weakness
- botulism.

Diagnosis

- Physical examination is non-specific.
- A dilated, gas-filled esophagus is often evident on survey radiographs of the neck.
- Contrast radiography of the esophagus can be used to confirm esophageal dilation. Gastric emptying time can be assessed concurrently.
- Barium swallow (fluoroscopy) is important to assess esophageal motility. The esophagus will be dilated and peristaltic waves weak or absent in the affected area.
- Thoracic radiographs should be taken to assess the lungs for secondary pneumonia.
- Poor esophageal motility and esophageal dilation are evident endoscopically. Esophageal ulceration may be observed secondary to gastroesophageal reflux. The stomach and duodenum should be evaluated for ulceration if possible.

Treatment

- A soft diet should be fed, consisting of mashes, slurries, or grass. It is important to ensure that a nutritionally balanced diet is provided. Unless grazing, foals should be fed at elbow-height.

Housed foals should be prevented from eating bedding because of the possibility of esophageal obstruction. This type of management may be required for months. Ideally, radiographs, fluoroscopy and/or endoscopy should be repeated (and normal) prior to introduction of solid feed.

- Metoclopramide (0.15 mg/kg SC q6 h) or bethanecol (0.3–0.4 mg/kg PO q6–8 h or 0.025–0.1 mg/kg SC q6–8 h) can be administered in an attempt to increase motility in the distal esophagus, increase distal esophageal sphincter tone and decrease gastroesophageal reflux. Broad-spectrum antimicrobial therapy is indicated if aspiration has occurred.
- The prognosis is guarded, particularly with severe and chronic aspiration pneumonia, but some foals may respond fully. The prognosis is likely best in cases where megaesophagus has developed secondary to gastric ulceration, and early, appropriate treatment is provided. In some congenital cases, esophageal function will improve over time.

Esophageal strictures (Figs 4.33–4.39)

History

Esophageal strictures may be congenital or acquired following esophageal injury. Congenital web strictures usually occur in the upper third of the cervical esophagus and are seen most frequently in Haflinger ponies, although they have been reported in other breeds.

Cases of acquired strictures usually have a history of severe or long-standing esophageal obstruction, esophagitis from gastroesophageal reflux, trauma (internal or external) or esophageal surgery. Circumferential esophageal lesions are more likely to cause strictures than linear lesions. Acquired strictures may occur in the thoracic or cervical esophagus. In young foals, clinical signs of stricture may not be apparent until solid feed is ingested.

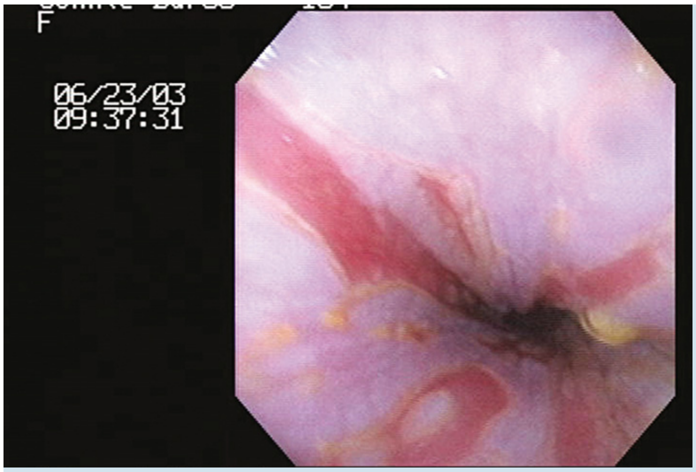


Figure 4.34: Endoscopic image of the esophagus demonstrating reflux esophagitis with marked ulceration of the esophageal mucosa.

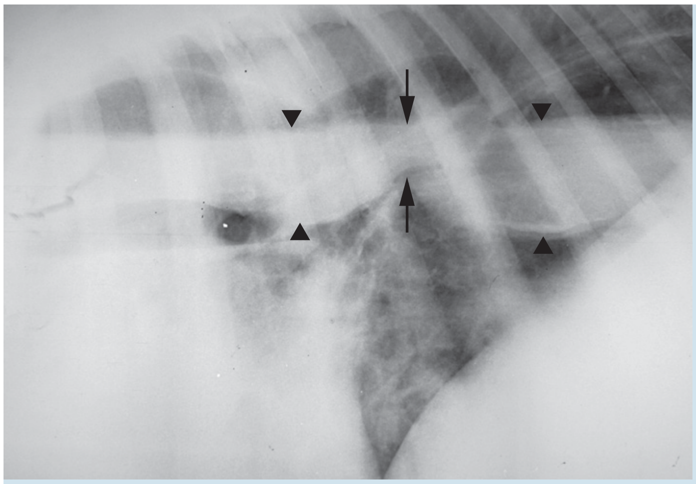


Figure 4.35: Thoracic radiograph from a foal that presented with a history of pneumonia. An esophageal stricture can be seen (arrows) in association with related areas of esophageal distension (arrowheads).

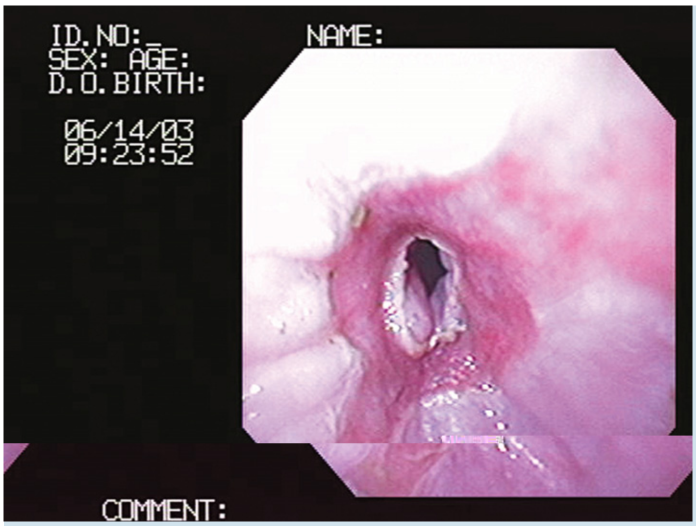


Figure 4.33: Endoscopic image of the esophagus demonstrating a circumferential stricture.

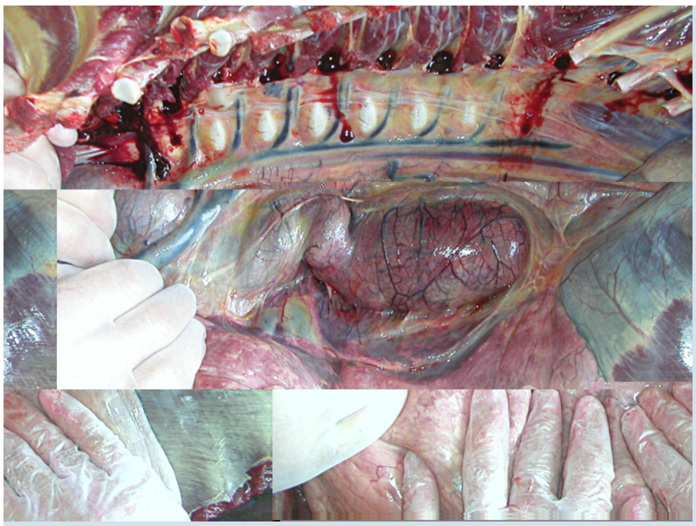
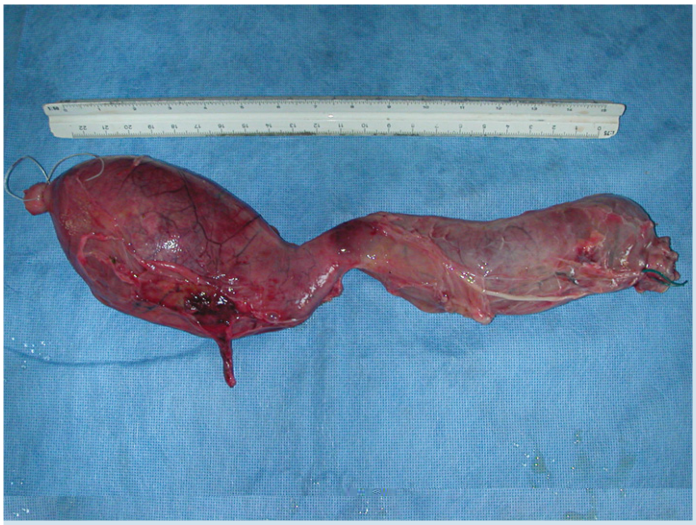


Figure 4.36: Postmortem image from the same foal as Fig 4.35 (head facing left). Again note the esophageal stricture and distension of the esophagus cranial and caudal to the lesion.



Figures 4.37 & 4.38: Section of the esophagus following removal. Note the marked fibrosis associated with the area of stricture.

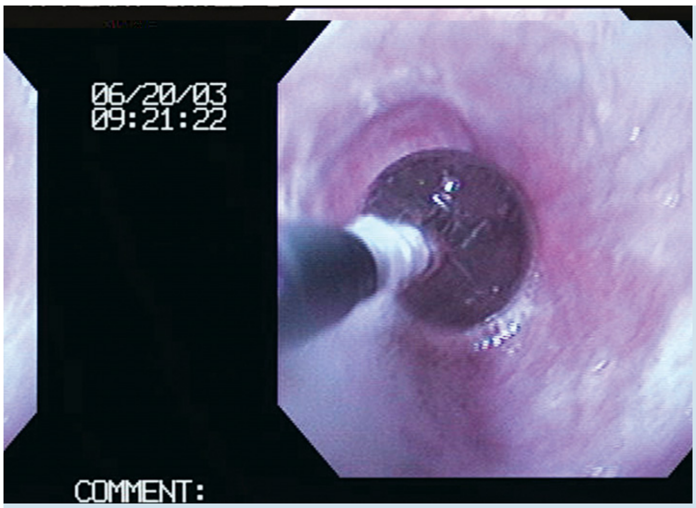


Figure 4.39: Endoscopic image of bougienage of an esophageal stricture.

Clinical signs

- Foals with congenital strictures may present with nasal reflux of milk shortly after birth.
- Recurrent esophageal obstruction with feed is the main clinical sign in older foals with acquired strictures. With chronic recurrent obstructions, megaesophagus may develop proximal to the site of stricture.
- When a stricture without obstruction is present foals can drink water and nurse normally.

Differential diagnosis

Recurrent esophageal obstruction may also be caused by:
- megaesophagus
- primary esophageal motility disorder
- pica
- cervical/mediastinal abscess
- persistent aortic arch.

Diagnosis

- History is important for identification of situations that may have led to stricture development.
- Esophageal dilation may be evident proximal to the stricture on survey radiographs.
- With contrast radiography, an area of narrowing of the esophagus may be identified; however it may be difficult to differentiate stricture from peristalsis. Repeated contrast radiographs can be useful for differentiation.
- Double contrast radiography can confirm the presence of a stricture.
- Endoscopic examination of the esophagus is important to visualize the stricture and identify concurrent problems (i.e. esophagitis, gastric ulceration).

Treatment

- The approach taken will depend on the severity of disease. In mild cases or in cases of congenital strictures dietary management may be adequate. Affected foals should be fed a soft diet consisting of mashes, slurries and/or grass. Some foals may never be able to tolerate hay or grass and must be fed a complete pelleted ration.
- Bougienage has been used to dilate esophageal strictures. It should be performed as early as possible as it will be less effective in older lesions. Surgical correction is reserved for cases that cannot be managed medically or with bougienage. However surgery can only be performed on cervical lesions and complications are common with a low success rate.

Pharyngeal paralysis (Figs 4.40 & 4.41)

History

Pharyngeal paralysis is a rare problem in foals that can have a variety of etiologies.

Figure 4.40: Milk evident at the nostrils of a foal with pharyngeal paralysis.

Figure 4.41: Esophagostomy in a foal with pharyngeal paralysis secondary to strangles infection.

- Pharyngeal paresis/paralysis has been seen in a number of foals with hypoxic ischemic encephalopathy.
- Any condition of the guttural pouch producing inflammation can affect pharyngeal function through irritation of cranial nerves running adjacent to the pouch.
- Trauma can also damage important cranial nerves.
- Equine herpes virus encephalomyelitis is a rare cause of pharyngeal paralysis, as is equine protozoal myelitis, and both are very uncommon in foals.
- Pharyngeal collapse or spasm has been associated with hyperkalemic periodic paralysis in Quarter horse foals.
- Idiopathic pharyngeal paresis in neonatal foals has been reported.

Clinical signs

- Dysphagia is the main clinical sign and is characterized by an inability to properly propel a food bolus from the oropharynx into the esophagus. Therefore, masticated food and saliva will be present at the nares and mouth.
- Aspiration pneumonia can develop secondarily.
- Dehydration, weight loss, poor body condition and weakness may also develop over time.
- Pharyngeal collapse can produce respiratory stridor. Exercise intolerance may be the only obvious sign in some cases. With more severe collapse, tachypnea or dyspnea may develop.

Differential diagnosis

Other causes of dysphagia include:

- cleft palate
- oral/pharyngeal trauma
- retropharyngeal abscessation
- esophageal obstruction
- other causes of cranial nerve damage/inflammation
- botulism.

Diagnosis

- A full neurological assessment, including evaluation of cranial nerves, should be performed.
- The head and neck should be carefully palpated for signs of malformation or trauma.
- The oral cavity should be examined for evidence of foreign bodies, abnormal odor, dental abnormalities, feed impaction and other lesions.
- Upper airway endoscopy is critical to evaluate the pharynx and larynx. Visual assessment of swallowing following spraying of a small volume of water in the pharynx is useful.
- Guttural pouch endoscopy should be performed to look for guttural pouch empyema or other conditions. Samples should be collected for bacterial culture and sensitivity testing if there is any evidence of infection or inflammation.
- Radiographs of the head can be useful to identify fractures secondary to trauma.
- Barium swallow (fluoroscopy) is another method to assess mastication and swallowing.
- Hyperkalemic periodic paralysis (HYPP) testing should be performed in Quarter horses or horses with Quarter horse lineage.
- Cerebrospinal fluid (CSF) analysis is indicated if the cause of pharyngeal paralysis has not been identified.

Treatment

- Nutritional support via nasogastric tube, esophagostomy or parenteral nutrition is often required.
- If guttural pouch infection is the cause, this should be treated with appropriate antimicrobials. Lavage of the guttural pouch is also useful if infection is present.
- Broad-spectrum antimicrobial therapy is required if aspiration pneumonia is present or aspiration has occurred.
- Systemic NSAID therapy (i.e. flunixin meglumine) is indicated in most cases. Tracheostomy may be required for emergency stabilization if dyspnea is present.
- Prognosis is variable depending on the inciting cause. In neonatal foals with idiopathic pharyngeal paresis, some foals will recover fully with short-term supportive care. The prognosis is guarded in most circumstances.

Choke (esophageal obstruction) (Figs 4.42 & 4.43)

History

Esophageal obstruction (choke) is an uncommon problem in foals. Most commonly, obstruction will be the result of ingestion of bedding, hay or other feed materials. Orphaned foals or those in a poorly enriched environment may be more prone to obstruction because of the increased likelihood of exploratory eating and pica. Rarely, a congenital lesion such as vascular ring anomaly, esophageal stenosis or esophageal cyst can cause esophageal obstruction.

Clinical signs

- The most common presenting complaint is the presence of bilateral nasal discharge containing feed material.
- Foals may cough while attempting to nurse and milk may stream from the nostrils after nursing. Feed material may be expelled during coughing and saliva may stream out of the mouth.
- If the obstruction is causing discomfort, the affected foal may stand with head and neck extended.
- Tracheal sounds are typically increased and fluidy. Aspiration pneumonia may be present in long standing or recurrent cases, and characterized by fever, variable cough, depression, weakness and increased lung sounds.

Differential diagnosis

Other causes of bilateral nasal discharge include:

- cleft palate in neonatal foals
- pharyngeal paresis/paralysis
- oral foreign body
- primary pneumonia
- megaesophagus
- botulism.

Diagnosis

- Occasionally, an obstruction or area of pain can be detected on palpation of the jugular furrow, particularly on the left side.
- A careful oral examination is important to rule out other problems. Food material may be present in the mouth.
- Inability to pass a nasogastric tube down the entire length of the esophagus is highly suggestive. If a very proximal obstruction is present, it may be difficult to differentiate difficulty in entering the esophagus from an obstruction.
- Endoscopy of the pharynx and esophagus is diagnostic. The trachea should be evaluated to assess the degree of aspiration.
- A radiodense obstruction may be evident radiographically. Barium should not be administered if an obstruction is suspected because of the potential for aspiration. Thoracic radiographs can identify concurrent aspiration pneumonia.

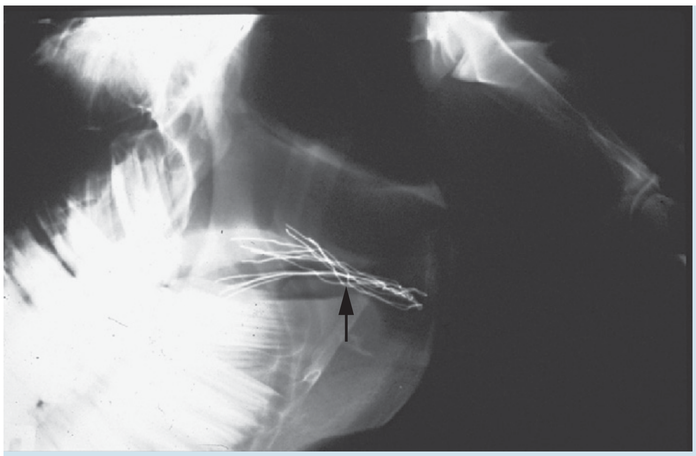


Figure 4.42: Radiograph of the head demonstrating a wire foreign body in the pharynx (arrow).

Figure 4.43: Ingesta at the nostrils of a foal with choke.

Treatment

- In some cases, restriction of feed and water for 12–24 hours may be adequate. Foals should be muzzled so that they cannot ingest any feed or bedding. Intravenous fluid therapy is important to correct/maintain hydration and electrolyte status, and judicious over-hydration may assist with softening of obstructed material. Nutritional support, ranging from supplementation of fluids with glucose to total parenteral nutrition, may be required depending on the duration of obstruction, age of the foal, overall health status and chosen treatment method.
- Gentle attempts to move the obstruction with a nasogastric tube can be successful in some cases, but care must be taken not to traumatize the esophagus. Administration of *N*-butylscopalammonium bromide (20 mg/dL) (Buscopan®) may decrease esophageal muscle spasm thus facilitating the passage of the nasogastric tube. Water can be flushed gently to help break up or move the obstruction, but care must be taken to avoid excessive pressure or aspiration. Mineral oil should never be used.
- A preferred approach involves placing a cuffed endotracheal tube in the esophagus, with a smaller tube inserted inside for flushing water. This method prevents aspiration and allows for large-volume lavage.
- Broad-spectrum antimicrobial therapy is indicated in virtually all cases because of the risk of aspiration pneumonia.
- If possible, a barium swallow (fluoroscopy) should be performed following resolution of the obstruction to assess esophageal motility. If fluoroscopy is not available, lateral radiographs of the neck following barium administration can be useful to detect esophageal dilation or pooling of barium. Feeding of solid foods should be restricted, preferably until normal motility is evident. Solid feed should be gradually introduced, initially in the form of gruels of pelleted feed or hay cubes, followed by soaked hay or grass.
- Esophageal stricture is a potential complication that is more common secondary to severe circumferential ulceration. Esophageal rupture carries a very poor prognosis. The prognosis is grave if a congenital disorder such as vascular ring anomaly is the cause because the problem may be difficult to diagnose and treat.

Gastroduodenal ulceration (Figs 4.44–4.54)

History

Gastric ulcers are common in foals, particularly those that are stressed or concurrently ill. Many foals with ulcers have no clinical signs and ulcers may be an incidental finding. In some cases, serious complications can develop. Foals with a history of ill-thrift, recent or ongoing disease, hospitalization, NSAID therapy, transportation, early weaning or management changes, and foals that have been orphaned, should be considered at higher risk for gastric ulcers.

Clinical signs

- Intermittent mild colic is the main clinical finding in those with clinical signs.
- Bruxism and lying on the back are particularly common.
- Diarrhea is also reported.

Figure 4.44: Colic in a foal with gastric ulceration. Such foals frequently prefer to lie on their backs.

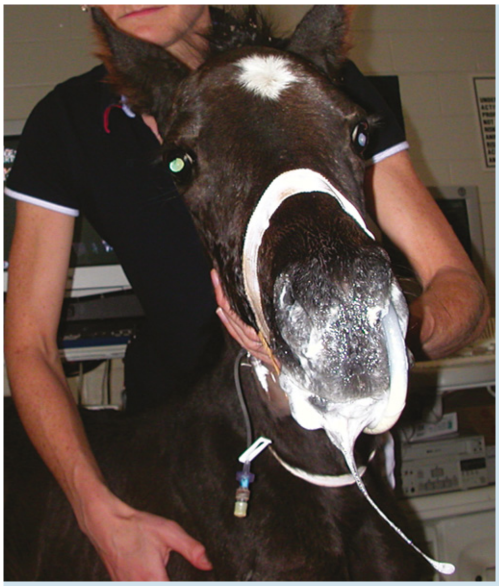


Figure 4.45: Bruxism and salivation in a foal with gastric ulceration and pyloric stenosis.

Differential diagnosis

Other causes of intermittent colic include:

- pyloric stenosis
- enteritis
- improper diet
- lactose intolerance
- abdominal abscessation
- gastrointestional impactions.

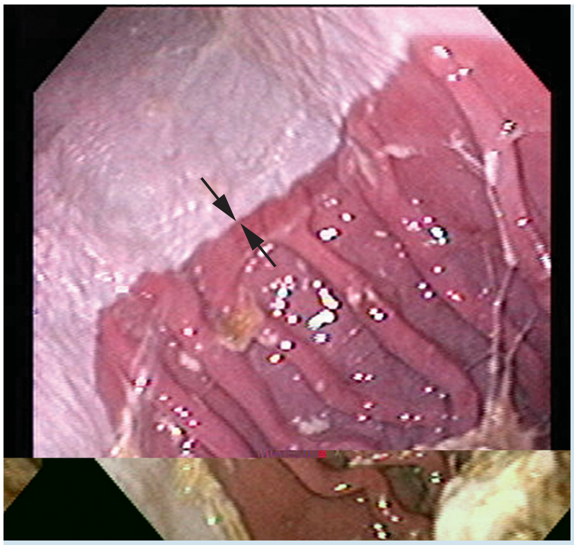


Figure 4.46: Endoscopic image of a normal stomach; note the margo plicatus (arrows) which is the junction between glandular and non-glandular/squamous regions.

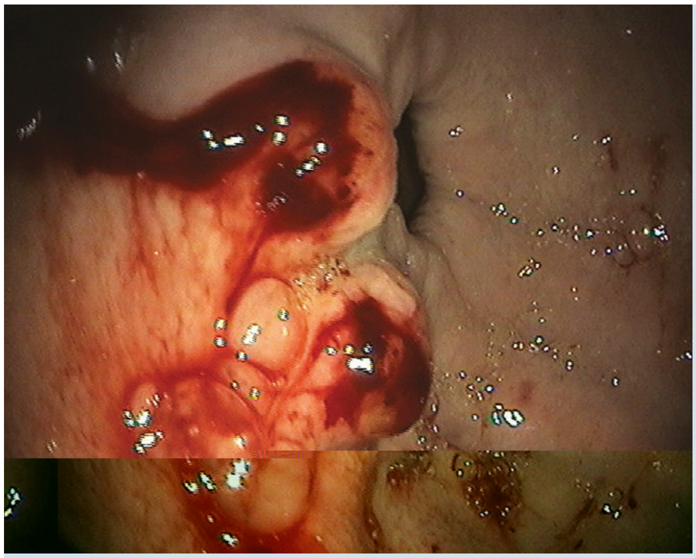


Figure 4.48: Endoscopic image of the stomach demonstrating bleeding ulcers of the glandular mucosa.

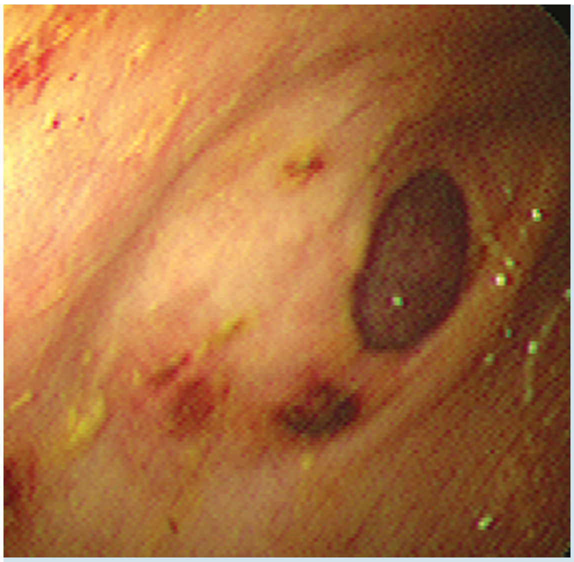


Figure 4.47: Endoscopic image of the stomach demonstrating ulceration of the non-glandular/squamous mucosa.

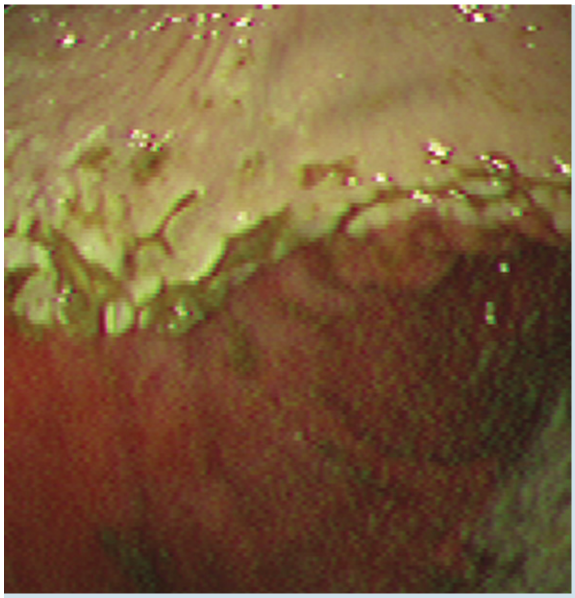


Figure 4.49: Endoscopic image of the stomach demonstrating ulceration along the margo plicatus.

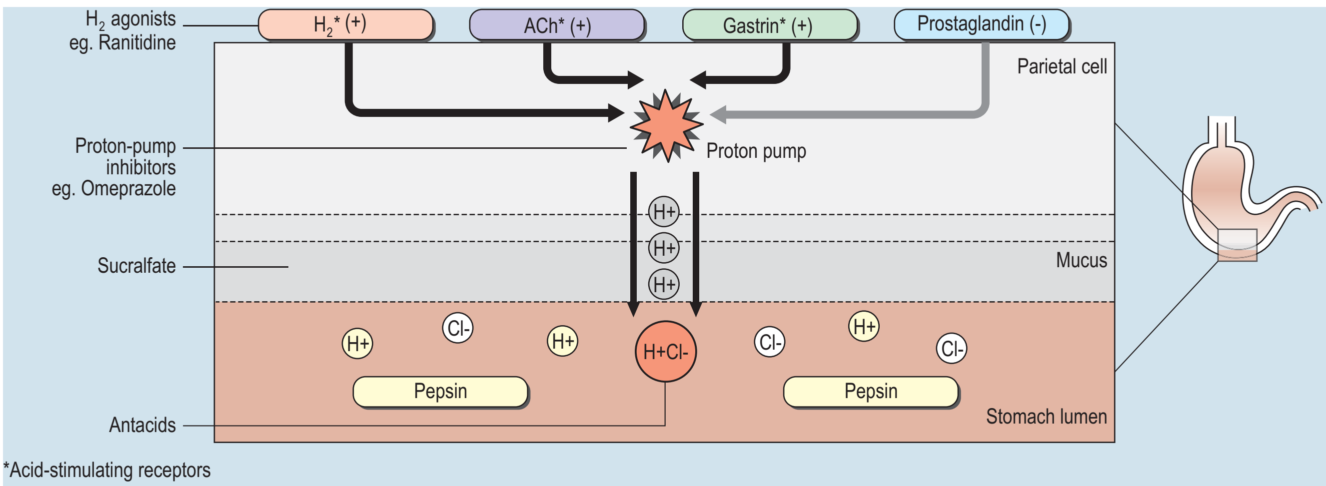


Figure 4.50: Schematic image of a gastric gland demonstrating the mode of action of omeprazole and ranitidine.

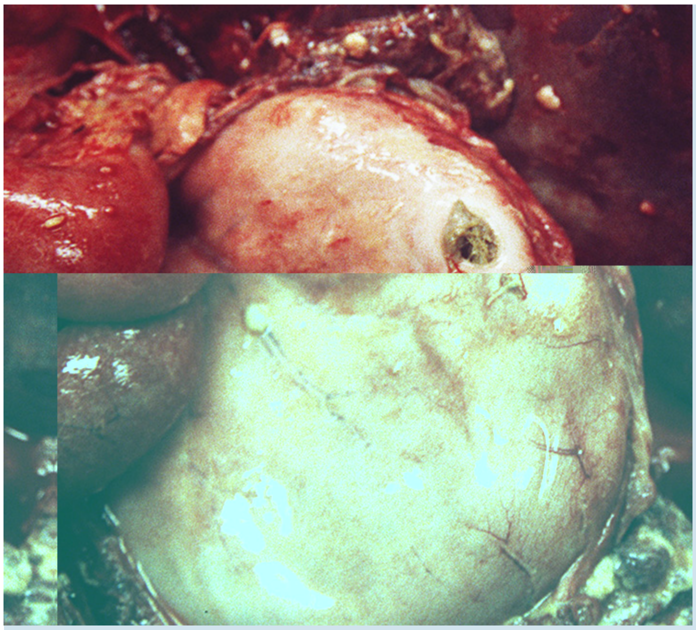


Figure 4.51: Postmortem image showing gastric perforation, which occurred secondary to severe ulceration.

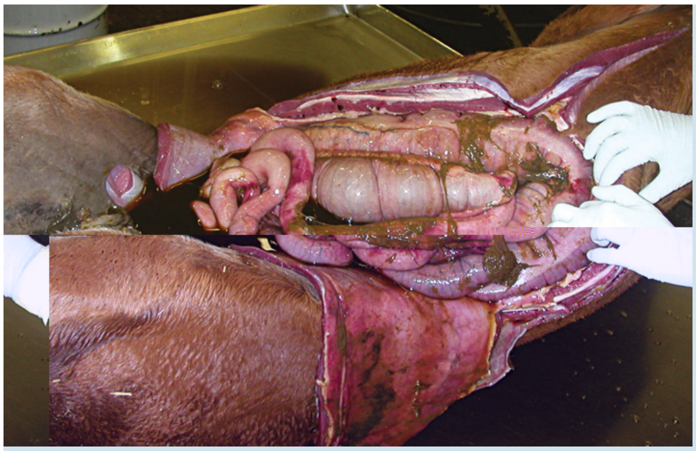


Figure 4.52: Postmortem image from a foal with gastric perforation showing marked abdominal contamination with ingesta.

Figure 4.53: Sudden onset abdominal distension and collapse in a 4-month-old foal with duodenal perforation.

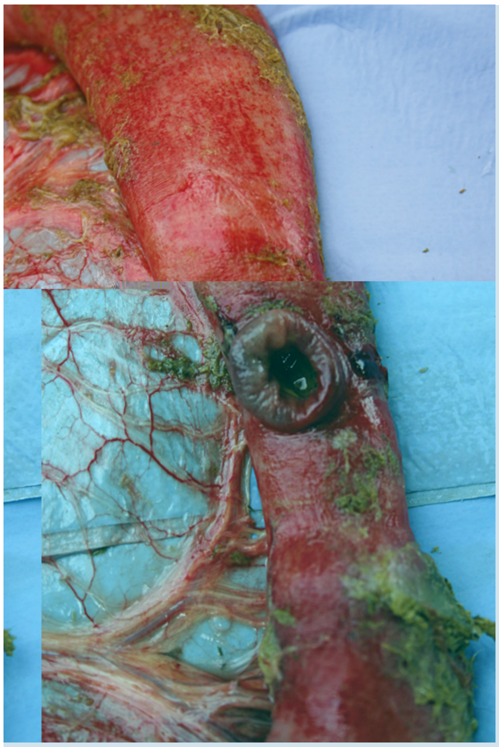


Figure 4.54: Same foal as in Fig 4.53. Note the well defined area of duodenal perforation. This foal had not demonstrated any signs consistent with gastroduodenal ulceration prior to perforation.

Diagnosis

- Gastroscopy is required for definitive diagnosis. Both the squamous mucosa and the glandular mucosa should be examined. Care should be taken not to over-interpret the clinical significance of mild ulceration. The duodenum should be evaluated concurrently.
- In some foals with duodenal ulceration, thickening of the duodenal wall may be evident via ultrasound (serosa >3.5 mm in thickness).
- Bleeding ulcers are uncommon and fecal occult blood is usually not detected even if significant bleeding is occurring.
- The use of sucrose permeability testing as an alternative diagnostic method has not yet been reported in foals.
- Pyloric stenosis should be considered in foals with chronic gastric reflux, or weight loss, that have a history of recent or ongoing gastric ulceration.

Treatment

- Omeprazole suppresses gastric acid secretion and is the best-proven anti-ulcer therapy in horses.
- Ranitidine is also widely used but less objective information is available.
- Sulcralfate is a disaccharide (sucrose) aluminum hydroxide complex. In acid environments sucrose is freed from the aluminum and binds

to anions of damaged GI epithelial cells forming a sticky viscous gel. Other properties such as increased blood flow via enhanced prostaglandin synthesis and binding of epidermal growth factor have been reported. These local effects may stimulate healing and are useful in the treatment of ulceration of the glandular mucosa or severe ulceration of the non-glandular/squamous mucosa.

- Stressors or other risk factors should be eliminated or reduced, if possible.
- With moderate to severe ulceration, a minimum of 3–4 weeks of treatment should be provided initially. Ideally, gastroscopy is repeated following the initial treatment course.
- Peritonitis from perforated gastric or duodenal ulcers is a concern; however clinical signs of gastric ulceration may not be present prior to perforation.
- Pyloric stenosis is an uncommon but serious sequel of severe gastroduodenal ulceration. Pancreatitis and cholangiohepatitis are other uncommon secondary conditions (see Chapter 10, p 282).
- Routine anti-ulcer treatment of high-risk (i.e. concurrently ill, hospitalized) foals is controversial. It is possible that gastric acid suppression could predispose foals to infectious diarrhea.

Pyloric stenosis (Figs 4.55–4.63)

History

Pyloric stenosis is a rare condition in foals that typically develops secondary to severe gastroduodenal ulceration. Congenital stenosis may also occur.

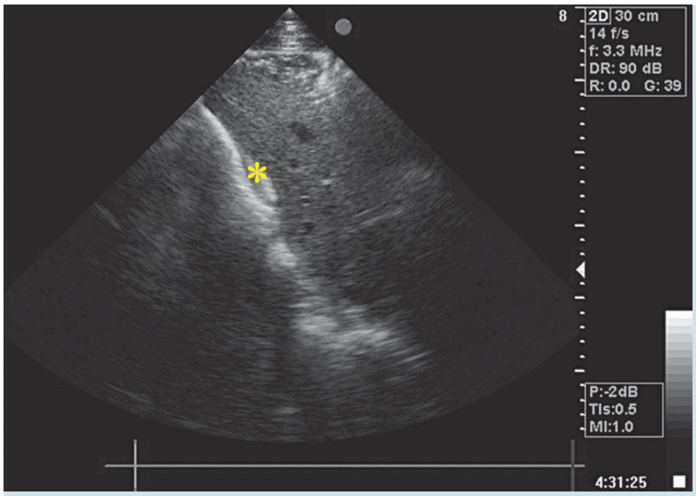


Figure 4.55: Ultrasonographic image of a normal closed duodenum (*) which can be visualized between the liver and colon on the right hand side of the abdomen (head facing right).

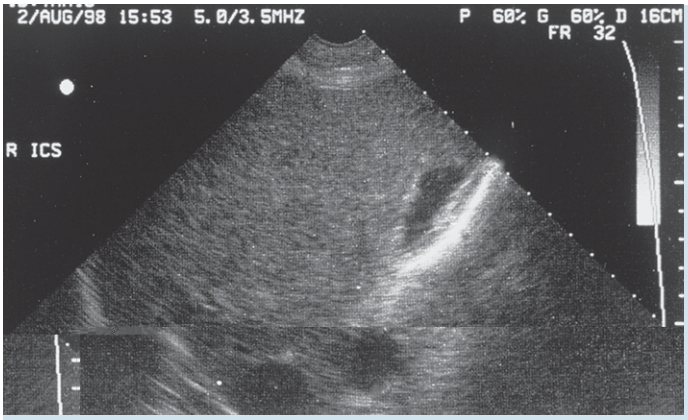


Figure 4.56: Ultrasonographic image of a normal open duodenum (*) (head facing left).

A B

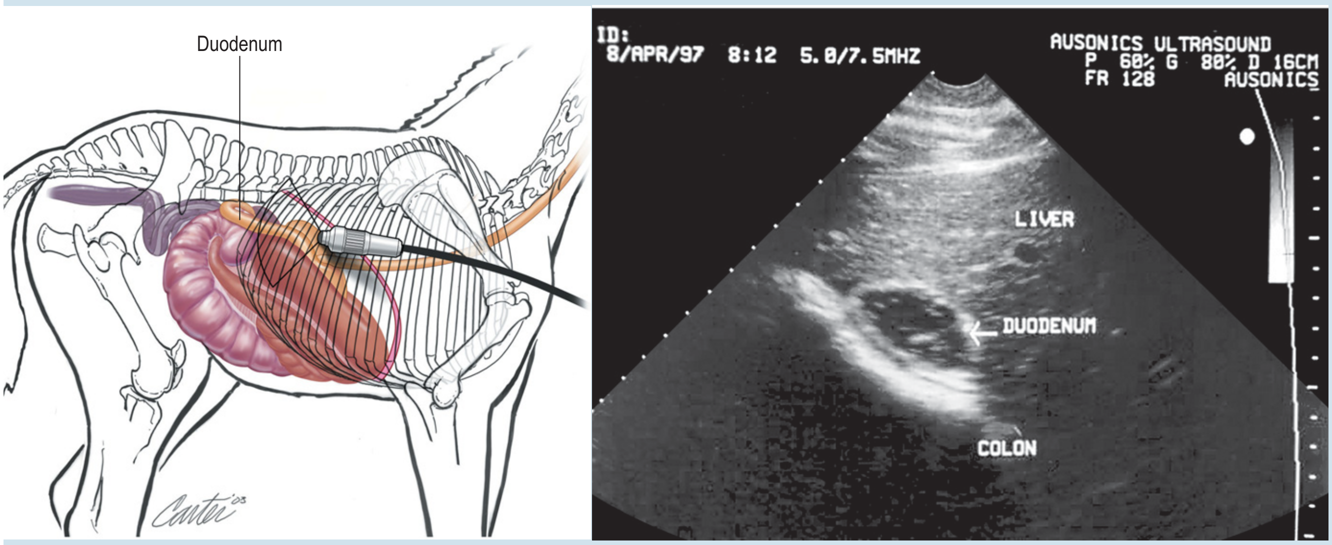


Figure 4.57: Schematic and ultrasonographic images of a distended duodenum found along the caudal border of the right lobe of the liver in the mid portion of the right abdomen (note in the schematic drawing the liver has been removed) (from Slovis NM 2003 Gastrointestinal failure. Clinical Techniques in Equine Practice 2:79–86).

Clinical signs

Intermittent colic, weight loss, ill-thrift, weakness and lethargy are common. Marked gastric distension with associated ileus and reflux may be seen, especially in more severe cases. The volume of reflux may be large (>10 L/day) despite appropriate prokinetic and other medical therapies. Signs of colic are variable but tend to be mild to moderate in severity.

Differential diagnosis

Other causes of low-grade, chronic colic and weight loss include:
- gastric ulceration
- duodenal ulceration

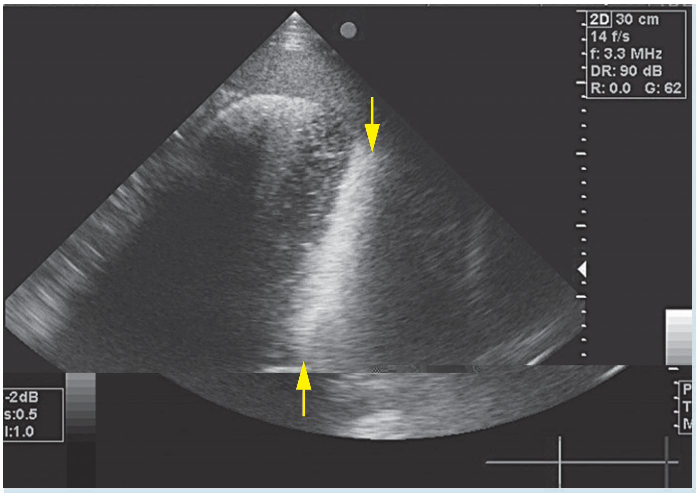


Figure 4.58: Ultrasonographic image of the left cranial abdomen demonstrating gastric distension with a gas fluid interface seen as a hyperechoic line (arrows). Gastric distension and fluid accumulation are commonly seen with pyloric stenosis.

- abdominal abscess
- esophagitis.

Diagnosis

- Severe gastric ulceration is usually evident endoscopically. This may be primary or secondary. Visualization of the pylorus may be difficult because of fluid accumulation within the stomach.
- Gastric emptying can be assessed via contrast radiography. Barium administered via nasogastric tube should be observed in the duodenum within 15 minutes, with the stomach essentially empty by $1–1^{1}/_{2}$ hours.
- Ultrasonographic examination of the abdomen may be unremarkable. Gastric distension is usually observed. Thickening of the duodenum (serosa >3.5 mm) may be present if significant duodenal ulceration and inflammation are present.
- Definitive diagnosis is made at surgery.

Treatment

- Medical therapy involves treatment of gastric ulceration and increasing gastric emptying. Gastric emptying can be stimulated by bethanecol or metoclopramide (see Appendix 5).
- Affected foals should be fed small meals frequently. Grass, pelleted rations or slurries are preferred as passage of these materials through the pylorus is easier than hay.
- Supportive therapy such as intravenous fluids, DMSO to decrease pyloric edema, analgesics, or parenteral nutrition may be required in some cases.
- Gastrojejunostomy or gastroduodenostomy may be required in severe cases. Reports of foals with long-term survival and normal growth rates following surgery are limited. In general if there is not a prompt response to medical therapy, prognosis is guarded.

A B

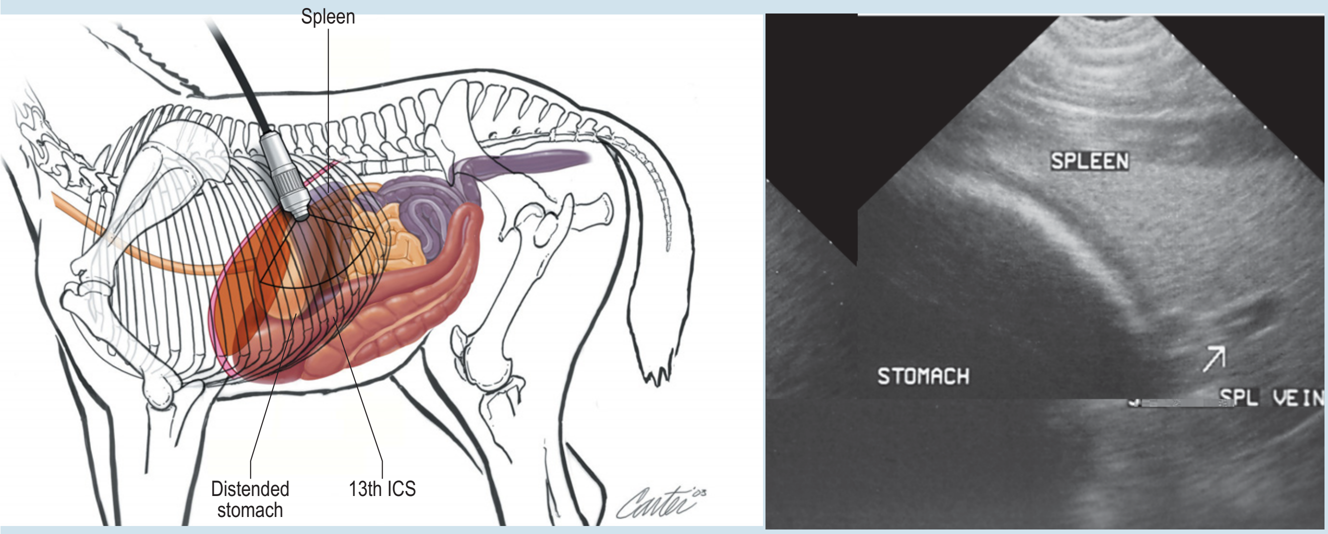


Figure 4.59: Schematic and ultrasonographic images of a normal foal's stomach visualized in the left 9th to 13th intercostal spaces, medial to the spleen (from Slovis NM 2003 Gastrointestinal failure. Clinical Techniques in Equine Practice 2:79–86).

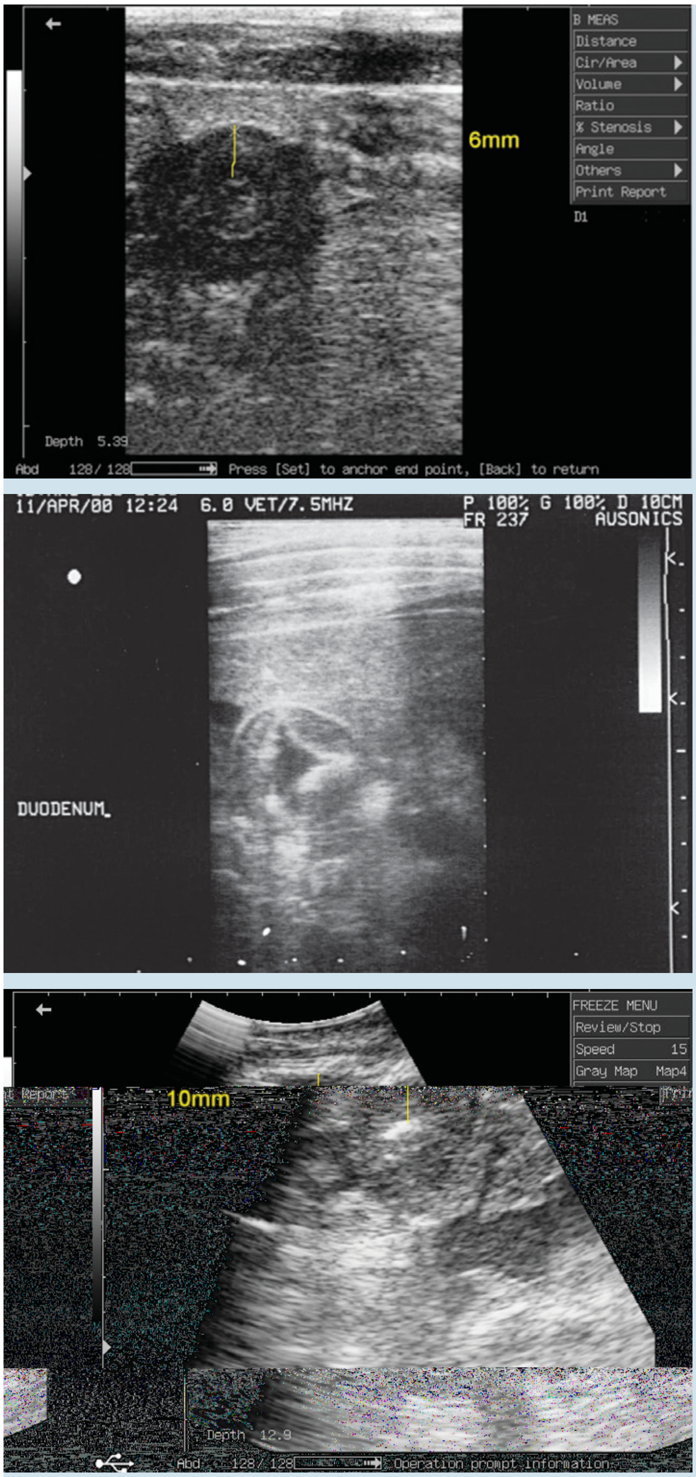
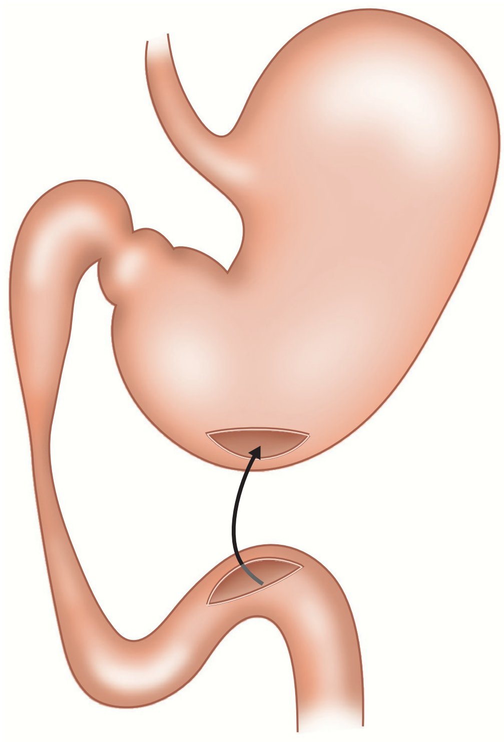
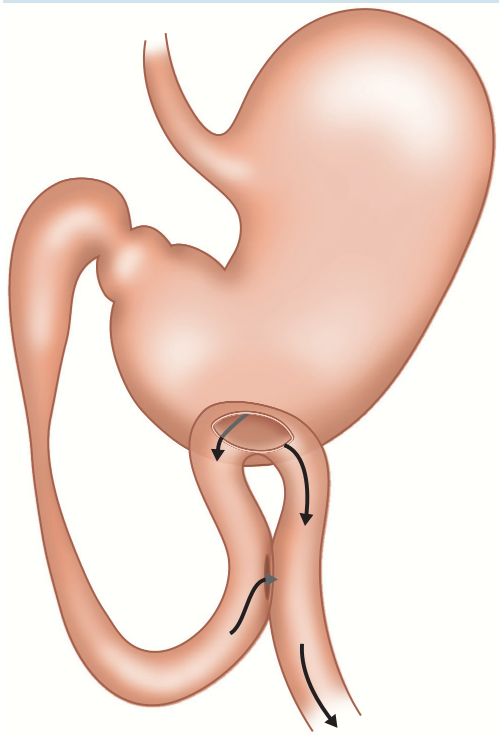


Figure 4.60–4.62: Ultrasonographic images of duodenal thickening in cross section. Normal serosal thickness is <3.5 mm.



(a)



(b)

Figure 4.63a&b: (a) Schematic representation of a gastrojejunostomy. (b) Schematic representation of a gastrojejunostomy and jejunojejunostomy; the latter is performed to allow contents of the duodenum and proximal jejunum to drain back into the jejunum.

Diarrhea

Diarrhea is the most common ailment of young foals and the number one reason for veterinary visitation. Diarrhea has a broad range of etiologies with a corresponding range of prognoses. In many instances the clinical signs may appear quite similar despite differing etiologies. Treatment in many cases of diarrhea is similar, with supportive care such as fluid therapy playing a large part in a successful outcome.

Differential diagnosis of diarrhea in foals by age group

0–10 days

Non-infectious diarrhea

- Foal heat diarrhea is a physiological event associated with development of normal large bowel function; foals usually remain bright and nurse normally.
- Diarrhea secondary to meconium impaction
- Errors of feeding; incorrect volume or frequency, especially in orphaned foals
- Other *infrequent* causes of diarrhea in this age group are congenital lactose intolerance, gastric ulceration and sand enteritis/colitis.

Infectious diarrhea

- Viral infections
  - rotavirus
  - coronavirus/adenovirus (usually seen in immunocompromised foals)
- Bacterial infection
  - Gram-positive enterocolitis: *Clostridium* spp
  - Gram-negative enterocolitis: *Escherichia coli*, *Salmonella* spp, *Actinobacillus* spp
- Fungal infection
  - *Candida*/*Mucor* spp (seen in immunocompromised foals)
- Protozoal infection
  - *Cryptosporidium* spp.

10 days to 6 weeks

Non-infectious diarrhea

- Foal heat diarrhea (normally seen up to 2 weeks of age)
- Dietary induced diarrhea
  - errors of feeding
  - post enteritis lactose intolerance
  - dietary hypersensitivity (rare)
  - copper deficiency (rare)
  - sand enteritis/colitis (rare)
- Antibiotic associated diarrhea – most commonly seen with orally administered antibiotics but can occur with parenteral administration
- Gastric ulceration.

Infectious diarrhea

- Viral infection
  - rotavirus
  - coronavirus
- Bacterial infection
  - Gram-positive enterocolitis: *Rhodococcus equi* (uncommon in foals less than 6 weeks but may occur), *Clostridium* spp
  - Gram-negative enterocolitis: *E. coli*, *Salmonella* spp, *Actinobacillus* spp, *Campylobacter* spp
- Fungal infection
  - *Candida*/*Mucor* spp (immunocompromised foals); this is the typical age group in which this type of diarrhea is seen in Severe Combined Immunodeficiency of Arabian foals.
- Protozoal infection
  - *Cryptosporidium* spp
- Parasitic infection
  - *Strongyloides westerii*, *Parascaris equorum*, *Strongylus vulgaris*.

6 weeks to 6 months

Non-infectious diarrhea

- Dietary induced diarrhea
  - errors of feeding
  - post enteritis lactose intolerance
  - dietary hypersensitivity (rare)
  - copper deficiency (rare)
  - sand enteritis/colitis (rare)
- Antibiotic associated diarrhea
- Gastric ulceration.

Infectious diarrhea

- Viral infection
  - rotavirus
  - coronavirus
- Bacterial infection
  - Gram-positive enterocolitis: *R. equi*, *Clostridium* spp, *Lawsonia intracellularis*
  - Gram-negative enterocolitis: *E. coli*, *Salmonella* spp, *Actinobacillus* spp, *Campylobacter* spp
- Fungal infection
  - *Candida*/*Mucor* spp (immunocompromised foals).
- Protozoal infection
  - *Cryptosporidium* spp
- Parasitic infection
  - *Strongyloides westerii*, *Parascaris equorum*, *Strongylus vulgaris*.

Non-infectious causes of diarrhea

Foal heat diarrhea (Fig 4.64)

Most foals experience a transient bout of diarrhea around 1 week of age, which may last 1–7 days and is usually seen at the time of the first post-foaling estrus. This is a normal physiological event and is thought to be due to the development of normal large bowel function.

Foal heat diarrhea is typified by a bright and active foal that continues to nurse well and experiences no episodes of fever. The consistency of the diarrhea may vary from pasty to watery. The diarrhea does not usually have a strong odor.

Rarely foals with watery diarrhea may develop signs of dehydration necessitating fluid therapy.

Should any fever, depression, decrease in appetite or malodorous diarrhea be noted further diagnostics should be performed as there are many other causes of diarrhea in this age group.

Diarrhea secondary to meconium impaction (Figs 4.65 & 4.66)

Diarrhea during or after resolution of meconium impaction is not uncommon; however it is typically mild and self-limiting with a dark brown colour. Sometimes diarrhea can be passed around an impaction. Diarrhea may develop as a result of treatments (particularly if osmotic cathartics are used), withholding of feed, or “stress”. Effects on development of the normal intestinal microflora may also result in idiopathic diarrhea or predispose to infectious diarrhea.

No abnormalities other than diarrhea are usually present. Occasionally, other signs such as dehydration, abdominal pain, depression, weakness and anorexia develop.

Figure 4.64: Foal heat diarrhea. Note the soiling of the hindquarters.

Diagnosis

- Mild diarrhea developing during or shortly after treatment of an impaction is used for diagnosis. If no other abnormalities are present, further testing may not be indicated.
- Fecal samples can be submitted for testing for enteropathogens.

Treatment

Treatment is usually not required and diarrhea will resolve within a few days. If signs in addition to diarrhea are present, supportive therapy and further diagnostic tests are indicated.

Figure 4.65: Normal meconium. Meconium may be present as discrete balls or may have a pasty consistency as shown.

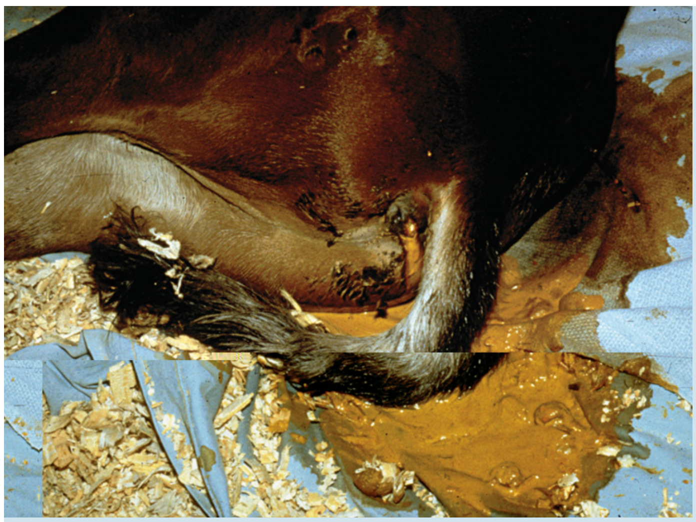


Figure 4.66: This foal has just received a soapy enema to aid in the passage of meconium. Note the discrete meconium balls that can be seen within the feces that have been passed.

Lactase deficiency

History

Also known as lactose intolerance, lactase deficiency can develop secondary to any insult affecting the lactase-producing cells in the small intestine. Infectious enteritis is usually the inciting cause, although ischemic damage could also occur. With lactase deficiency, lactose is not digested and passes intact into the cecum and large intestine, where it acts as a substrate for bacteria, resulting in gas production and osmotic diarrhea.

Clinical signs

- If lactase deficiency is the sole problem, as would be the case where infectious enteritis has resolved, foals are bright and alert with chronic diarrhea.
- Bloating and intermittent abdominal pain are common.
- Varying degrees of depression, weakness, weight loss and dehydration may develop.
- In severe cases, signs attributable to severe hypoglycemia (profound weakness, hypothermia, seizures) may ensue.
- Lactase deficiency may also occur concurrently with infectious enteritis. In these situations, most clinical signs are attributable to the primary disease process.

Differential diagnosis

See pp. 101, 102.

Diagnosis

- Lactose tolerance test. Following a 4-hour fast, take a baseline blood glucose sample then administer 1 g/kg lactose as a 20% solution via nasogastric tube or bottle. Repeat blood glucose level at 30-minute intervals for 3 hours. An increase in blood glucose of <2 mmol/L indicates lactase deficiency or glucose malabsorption. To rule out glucose malabsorption, the test can be repeated using glucose instead of lactose.
- Response to treatment can be supportive of a diagnosis of lactase deficiency.
- Testing for other enteropathogens is indicated as described elsewhere.
- Blood glucose level should be evaluated and monitored during the treatment period in foals with clinical signs of hypoglycemia.

Treatment

- Supportive therapy is important. Intravenous fluid therapy may be required in dehydrated foals. If clinically-relevant hypoglycemia is present, supplementation of intravenous fluids with glucose may be required initially.
- Any underlying disease process should be treated if possible.
- Specific treatment involves oral administration of lactase (1500–3000 IU q4–12 h). Frequency of administration depends on the severity of disease. The duration of treatment depends on the severity of damage to lactase-producing cells and whether a disease process is ongoing. Typically, 1–2 weeks of treatment is adequate. Lactase administration can be tapered and clinical response noted to help determine when to cease supplementation. Weaning of older foals should be considered if lactase administration is problematic and the foal is clinically stable.

Sand enteritis / colitis (Figs 4.67 & 4.68)

History

Chronic ingestion of sand can result in physical irritation of the bowel and enteritis. It is more common in certain regions and almost exclusively affects animals that spend a large percentage of time on sandy pastures, dry lots, arenas or stalls with a sand base. In these

Figure 4.67: Foal with sand colitis. Note the sand particles evident on the hindquarters.

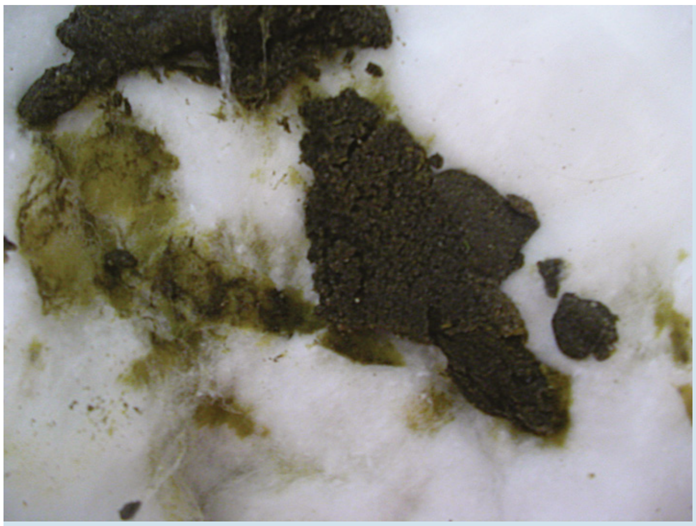


Figure 4.68: Feces from the foal in Fig 4.67. The feces consist almost entirely of sand.

areas it is a common cause of diarrhea and colic in foals of all ages.

Clinical signs

- Diarrhea is the main presenting sign.
- Colic, weight loss, intermittent pyrexia and ill-thrift may also be present.
- Signs of toxemia or cardiovascular compromise that can accompany diarrhea of other etiologies are uncommon unless the intestinal mucosa has been significantly damaged.
- Intestinal rupture is rare, but may occur and be accompanied by signs of septic peritonitis.

Differential diagnosis

See pp. 101, 102.

Diagnosis

- Sand-induced diarrhea is rarely accompanied by dehydration or signs of cardiovascular compromise. Affected foals are usually bright and nurse normally.
- Fecal samples are negative for common enteropathogens, including *Salmonella* spp, rotavirus, *Clostridium difficile* and *C. perfringens*.
- Identification of large quantities of sand in feces is suggestive, more so if other causes of diarrhea have been ruled out. Fecal sand can be evaluated by mixing feces and water in a rectal sleeve and looking at the volume of sand that settles at the bottom.
- Abdominal radiography can be used to identify sand accumulation in the intestinal tract.
- Hematology and peritoneal fluid are unremarkable unless intestinal compromise or rupture has developed.

Treatment

- Mild cases may recover with minimal treatment following removal from the sand source.
- In more severely affected cases supportive care, particularly intravenous fluid therapy, may be required. Anti-inflammatories are useful for decreasing bowel inflammation.
- While the efficacy of bulk laxatives is controversial, psyllium (0.5–1 g/100 kg PO q6–24 h) is often used. Duration of psyllium therapy should be based on repeated fecal sand analysis and/or repeated abdominal radiographs. Intermittent psyllium treatment may be indicated in foals on high-risk farms.
- Where possible management should be addressed to decrease sand exposure.

Feeding errors

Feeding errors are most commonly seen in hand-reared foals fed powdered milk. Errors may be associated with:

- incorrect volume for size of foal
- incorrect concentration of formula
- incorrect temperature
- incorrect mixing
- feeding of left-over milk at next feeding.

Careful attention should be given to feeding of hand-reared foals especially at a young age as many may also have experienced some degree of failure of passive transfer and thus be more susceptible to enteric infections.

Hand-reared foals are also more stressed and have a higher tendency to develop intestinal problems, especially gastric ulceration.

Other foals may develop feed associated diarrhea associated with long periods of confinement, separation from the mare, or abnormal dietary composition.

Antibiotic-induced diarrhea (Figs 4.69 & 4.70)

History

Antibiotic-induced diarrhea is most commonly related to the administration of oral antibiotics but can be caused by parenteral administration. The antibiotics most frequently associated with this type of diarrhea are:

- neomycin
- lincomycin
- members of the macrolide family (erythromycin, azithromycin and clarithromycin)
- chloramphenicol.

Oxytetracycline and other tetracyclines are classically stated as frequently causing diarrhea and while this may be the case in many adults and older foals, it does not appear to be the case in young foals.

Clinical signs

- Diarrhea frequently occurs soon after the commencement of antibiotic treatment but in some cases may not occur until well into the course of treatment or soon after treatment has ceased.
- Diarrhea is moderate to severe, with failure to withdraw the inciting cause resulting in progressively worsening diarrhea and associated clinical signs.
- Foals are usually non-febrile but progression may result in signs of dehydration, cardiovascular compromise and endotoxemia.

Diagnosis

Antibiotic-induced diarrhea should be suspected in any foal that suddenly develops diarrhea while on antibiotic treatment, especially if the original reason for antibiotic treatment was not related to the gastrointestinal tract, e.g. foal being treated for *R. equi* pneumonia that suddenly develops diarrhea.

Other causes of diarrhea, especially feeding changes that may be associated with confinement that coincides with treatment, should be considered.

Treatment

- Immediate cessation of antibiotic treatment is essential in any foal that suddenly develops diarrhea. In cases in which it is essential to maintain antibiotic treatment, e.g. septic arthritis, the use of antibiotics such as aminoglycosides that are not excreted or secreted into the bowel could be maintained.
- Diarrhea usually resolves quickly when the inciting cause is removed. However, if treatment with the offending antibiotic is

continued after diarrhea has commenced, it may become persistent and difficult to treat.

- In cases in which diarrhea does not resolve following removal of the suspected cause, further diagnostics should be performed to rule out other possible causes e.g. *Clostridium difficile*.
- Various treatments have been tried in cases that are slow to resolve. Probiotics are widely used and while there is little scientific evidence of their benefits many practitioners feel that they have a role in restoring some of the normal gut flora. Live yoghurt is also widely used based on the same principle but its efficacy is unproven.
- Metronidazole (10–15 mg/kg PO q8–12 h) is often used.
- Transfaunation can be attempted in long-standing cases that have not responded to other treatments. Fresh cecal contents are collected from a recently dead horse and are then passed through a fine gauge sieve – 1 L of the resultant fluid is administered in two equal doses of 500 mL, 4 hours apart by nasogastric tube. There are few reports of successful treatments in this manner and this procedure does carry a risk of infection.

Figures 4.69 & 4.70: Abdominal distension and diarrhea in a foal that was receiving macrolide antibiotics.

Infectious causes of diarrhea

Rotaviral diarrhea (Fig 4.71)

History

Rotavirus is a common cause of diarrhea in foals less than 3–6 months of age. Foals as young as 1 day of age may be affected. Outbreaks are not uncommon, particularly on large breeding farms. Other foals, adults or the environment may be sources of infection.

Clinical signs

- Diarrhea is the main complaint.
- Mild depression, partial anorexia and pyrexia are usually present, and may develop 24 hours before diarrhea is passed.
- Clinical signs may worsen as dehydration develops.
- Ileus resulting in abdominal distension is frequently seen.

Differential diagnosis

See pp. 101, 102.

Diagnosis

- ELISA, latex agglutination test or electron microscopy can be used to detect rotavirus in feces.
- Because of the potential for co-infection, testing for other enteropathogens should also be performed.
- A complete blood cell count, serum biochemical profile and plasma fibrinogen are useful to direct supportive therapy. IgG determination should be performed in foals less than 1 week of age.

Figure 4.71: Watery diarrhea in a foal with rotavirus infection.

- Translocation of bacteria is not uncommon in foals affected with rotavirus. Blood culture should be performed in moderate to severely affected foals to rule out secondary bacteremia.

Treatment

- Rotaviral diarrhea may be self-limiting and typically resolves within 3–5 days.
- Supportive therapy including intravenous fluid therapy and nutritional support may be required.
- Lactase supplementation may be useful because rotaviral diarrhea has been associated with lactase deficiency in other species.
- Affected foals and their dams should be regarded as infectious and appropriate measures should be taken to prevent spread to other foals.
- If ileus and abdominal distension are present, the use of prokinetics such as neostigmine and analgesics may be indicated (see p. 131).
- Phenolic disinfectants should be used for footbaths and stall cleaning as chlorine containing compounds are not effective against rotavirus.

Clostridial diarrhea (Figs 4.72–4.77)

History

Clostridium difficile and *C. perfringens* are the most commonly identified causes of diarrhea in foals in some areas. Outbreaks can occur and disease can range from mild to fatal. Foals as young as 12 hours of age may be affected. Clostridial diarrhea may be more commonly associated with preceding antimicrobial therapy.

Clinical signs

- Clinical signs can be highly variable. Diarrhea is the most consistent finding, but may not be observed initially in some cases. Bloody diarrhea may be passed; however fecal character has no diagnostic value.
- Varying degrees of abdominal pain, abdominal distension, dehydration, toxemia and weakness are present. Occasionally, severe abdominal pain and abdominal distension with small intestinal distension and hypomotility may be present, mimicking a strangulating small intestinal lesion.

Differential diagnosis

Diarrhea may also be caused by:
- salmonellosis
- rotavirus
- nutritional causes
- lactase deficiency.

Abdominal pain and distension may also be present with:
- other causes of diarrhea
- surgical intestinal lesion
- ruptured bladder.

Diagnosis

Clostridium difficile

- Toxin ELISA on feces. Ideally, the test should be able to detect both *C. difficile* toxins A and B, although tests that only detect toxin A are still useful. A single sample is adequate unless the sample was collected very early in disease.
- Fecal culture is less rewarding because *C. difficile* is difficult to isolate, and isolation of the organism does not necessarily indicate disease.
- Detection of glutamate dehydrogenase ("common antigen") in feces can be used in conjunction with toxin ELISA, but alone is not adequate for diagnosis.

Figures 4.72 & 4.73: Bloody feces in a foal with clostridial diarrhea. Such a large amount of blood is uncommon.

Figure 4.74: "Milk face" on a foal with diarrhea. This is commonly seen in foals that assume the nursing position, thereby prompting milk letdown by the mare and then fail to nurse. The milk then becomes stuck to the hair of the face or forehead.

Figure 4.75: Mare running milk. Udder distension and running milk may be the first signs of a foal's inappetance.

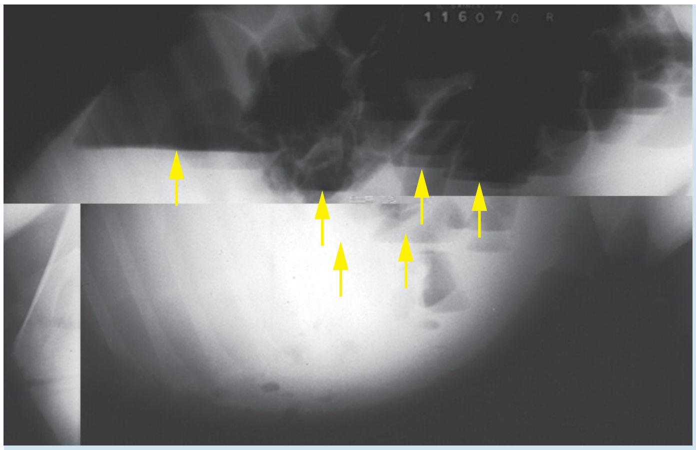


Figure 4.76: Radiograph from a foal with clostridial diarrhea (head facing left) demonstrating a large amount of fluid within the large intestine. The gas–fluid interfaces can be seen as a series of well defined horizontal lines (arrows).

Clostridium perfringens

- ELISA for *C. perfringens* enterotoxin. Currently, tests for other toxins are not available.
- Fecal culture is of limited use by itself because *C. perfringens* is very commonly found in normal foals. Culture followed by genotyping of isolates may be more useful, particularly if a Type C strain is identified. The relevance of the presence of other strains in the intestinal tract is unclear.

If severe abdominal pain and/or abdominal distension are present, abdominal ultrasonography and radiography are indicated. Intestinal rupture can develop secondary to enteritis so any deterioration in clinical condition should prompt a thorough re-evaluation.

Other diagnostic testing is important to direct supportive care. This should include a complete blood cell count, serum biochemical profile, plasma fibrinogen assay and blood culture.

Treatment

- Supportive therapy is critical. Intravenous fluid therapy is often required and oncotic support in the form of plasma or synthetic colloids may be necessary. Plasma is also required if failure of passive transport is present.

Figure 4.77: Small intestine from a foal with clostridial diarrhea. Abdominal pain was so marked in this foal that an exploratory celiotomy was performed.

- NSAID therapy may be useful and other analgesics or prokinetics may also be required (see p. 131).
- In neonatal foals with diarrhea, broad-spectrum antimicrobial therapy, such as a combination of penicillin and amikacin, is justifiable based on concerns about bacterial translocation and bacteremia.
- Gastric ulcer prophylaxis is controversial in neonatal foals and some authors recommend avoiding it because the increased gastric pH could allow for increased survival of ingested clostridia. Decisions regarding gastric ulcer prophylaxis should take into consideration such factors as the prevalence of clostridial infections in the area, concurrent illnesses, use of NSAIDs and other risk factors for the development of gastric ulceration (see p. 96). Sucralfate is a treatment for gastric ulceration that does not result in an increased gastric pH and may be a good choice in neonates at risk of clostridial infection (see pp. 98–99).
- Metronidazole appears to be effective against enteric clostridia and is frequently used.
- Di-tri-octahedral smectite (BioSponge™) may adsorb clostridial toxins in the intestinal tract.
- In some cases, severe abdominal pain, abdominal distension and small intestinal hypomotility will result in exploratory surgery because of concerns regarding a surgical lesion.
- Affected foals (and their dams) should be considered infectious.

Salmonellosis (Figs 4.78–4.80)

History

Salmonellosis is a cause of enteritis and sepsis in foals as young as 1 day of age. A small percentage of normal horses will transiently shed *Salmonella* spp. Foals are more susceptible to infection, and failure of passive transfer of maternal antibodies likely increases the chance of sepsis associated with salmonellosis.

Clinical signs

- Varying degrees of diarrhea, depression, weakness, dehydration, endotoxemia, hypotension, anorexia and pyrexia may be present.

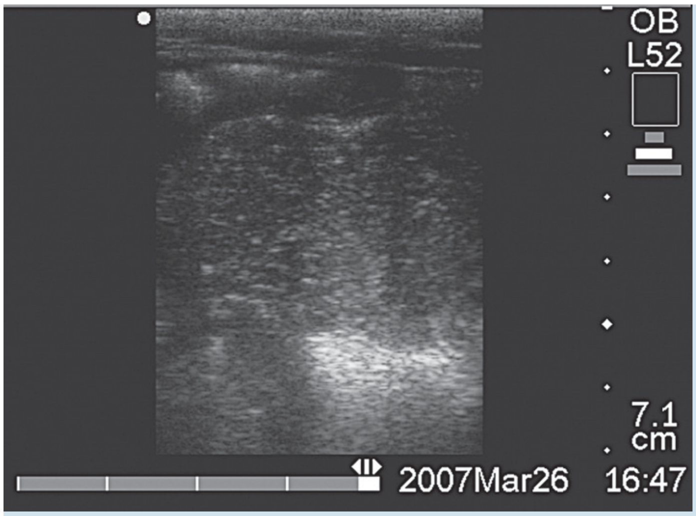


Figure 4.78: Ultrasonogram of the colon of a foal demonstrating fluid contents which were hypermotile in real time.

Figure 4.79: Watery diarrhea in a foal with salmonellosis.

- A common finding in foals with salmonellosis is severe leukopenia which is followed by a massive rebound leucocytosis.
- Severely affected foals may be recumbent from severe dehydration, toxemia, acidosis and/or hypoglycemia.
- Sepsis can occur secondary to salmonellosis, complicating the clinical presentation.

Differential diagnosis

Other causes of diarrhea include:

- *Clostridium difficile*
- *Clostridium perfringens*
- rotavirus
- lactase deficiency
- nutritional.

Severe cases may need to be differentiated from:

- peritonitis
- sepsis
- intestinal rupture.

Figure 4.80: Osteomyelitis in a foal with salmonellosis. Note the prominent lytic areas in the physis and diaphysis (arrows).

Diagnosis

- Fecal culture: Salmonella can be difficult to isolate and requires adequate sample size (minimum 10 g) and enrichment culture. Failure to isolate the organism even after a number of attempts does not rule it out. Although challenging, fecal cultures should be performed on feces from both the foal and dam as isolation of the organism provides a definitive diagnosis and antibiotic sensitivity can guide treatment.
- Polymerase chain reaction (PCR) test kits are available and may be useful in cases in which fecal cultures are negative.
- Complete blood cell count, serum biochemical profile and plasma fibrinogen for baseline information.
- IgG level should be evaluated in foals less than 2 weeks of age.
- Blood culture should be performed in foals with signs of systemic compromise.
- Testing for other enteropathogens should also be performed.

Treatment

- Broad-spectrum antimicrobial therapy is indicated in young foals, not to treat enteric *Salmonella* but for treatment or prevention of bacterial translocation. A combination of penicillin and amikacin is a reasonable initial choice, pending culture and sensitivity results.
- Intravenous fluid therapy is indicated in dehydrated, toxemic or hypotensive foals.
- Low-dose ("anti-endotoxin") doses of flunixin meglumine may be useful.
- Oral or nasogastric administration of di-tri-octahedral smectite (BioSponge™) may bind to intestinal endotoxin.
- Nutritional support may be required if foals are unable to adequately nurse (see: Feeding the sick foal, p. 129).
- J-5 plasma transfusion is useful in any affected foal and is critical in foals with failure of passive transfer of maternal antibodies (see

p. 297). Sepsis can also alter the distribution and catabolism of IgG making transfusions necessary in foals that had previously been assessed with adequate IgG. Synthetic colloids may also be given for oncotic support, if required.
- Foals should be monitored closely for signs of extraintestinal disease (i.e. septic arthritis).
- Infected animals should be regarded as infectious.

Colibacillosis

History

The exact role of *E. coli* in enteric infections in foals is debatable. Identification of *E. coli* in feces is not indicative of a primary enteric infection. However, blood cultures from septicemic foals frequently yield *E. coli* spp, and septicemic invasion may be more important than infection with the enteric or enterotoxigenic forms. Enteric infections may occur secondary to antibiotic usage or follow other infections.

Clinical signs

Diarrhea can range from mild to severe. Foals that develop diarrhea associated with *E. coli* septicemia are typically seriously ill, with marked fevers, depression and signs of endotoxemia.

Diagnosis

- As stated above the isolation of *E. coli* from fecal samples is inconclusive.
- Identification of specific enterotoxins is required for a definitive diagnosis of a primary *E. coli* enteric infection. Such testing has only a limited availability.
- Identification of *E. coli* in blood cultures is definitive for septicemia.

Treatment

Aggressive treatment of septicemic cases is required with antimicrobial selection based on sensitivity testing. Other important aspects of treatment include fluid therapy, correction of electrolyte and acid–base abnormalities, anti-endotoxin therapy, plasma transfusions for passive transfer of maternal antibodies and oncotic support.

Parasitism (Figs 4.81–4.83)

History

A variety of intestinal parasites can be found in foals but most are of limited clinical significance, even with large burdens.
- Ascarids (*Parascaris equorum*) are a significant concern, and are the most important intestinal parasite of foals in most areas.
- *Strongyloides westerii* may be a cause of disease in 2–4-week-old foals with significant worm burdens; however its true role in disease is of debate.
- Cyathostominosis (larval cyathostomiasis) may occur in foals as young as 3–4 months of age, but is more common in yearlings and older horses.
- *Cryptosporidium* and *Giardia* spp are uncommon causes of diarrhea, and are likely more important in immunocompromised foals.

Clinical signs

- Ill-thrift, weight loss, failure to grow normally and diarrhea are common presenting complaints.
- Intestinal obstruction and rupture with ascarids can develop particularly after deworming foals with a large burden.
- Significant hypoproteinemia and weight loss can occur with cyathostominosis.

Differential diagnosis

For other causes of diarrhea see above. Hypoproteinemia can also be caused by:
- salmonellosis
- clostridial diarrhea
- protein-losing nephropathy
- *Lawsonia intracellularis* infection
- poor diet.

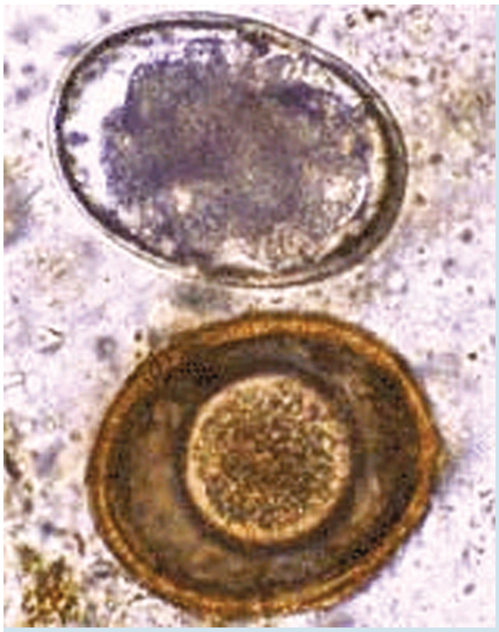


Figure 4.81: Strongyle egg (top) and ascarid egg (bottom).

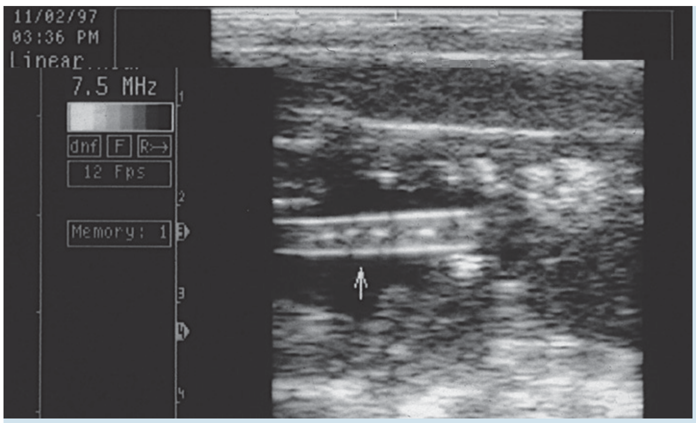


Figure 4.82: Ultrasonographic image of the ventral abdomen demonstrating the presence of a parasite (arrow) free within the small intestine.

Figure 4.83: Ileal impaction with ascarids. A small incision has been made through which large numbers of worms are passed.

Diagnosis

- Fecal flotation and smear can be used to identify certain parasites. Quantitation of fecal egg numbers can be useful. *However, ascarids and cyathostomes cannot always be identified in this manner.*
- Foals with severe ascarid infection may be seen to pass whole worms in their feces.
- In some cases of severe infection the parasites have been visualized within the bowel or free in the abdomen (in cases of intestinal rupture) by ultrasonographic examination.
- Testing for other enteropathogens, particularly those that are contagious, should be performed if diarrhea is present.
- Definitive diagnosis of certain conditions such as cyathostominosis is difficult and may only be made at surgery or necropsy.

Treatment

- Deworming should be addressed both at the individual and farm levels.
- Deworming programs are variable and depend on a variety of factors including geographic location (especially climate), amount of horse movement, horse density, pasture availability, pasture management, prevalence of individual parasites in the area and anthelmintic resistance.
- Excessive administration of dewormers should be avoided.
- Monitoring of fecal egg counts can be used to guide anthelmintic therapy, depending on the parasites of concern. Drugs with a narrow margin of safety (e.g. avermectins/milbemycin) should be used carefully and avoided in foals less than 12 weeks of age as they have been known to cross the blood–brain barrier in young foals leading to serious side effects.
- Deworming programs should pay particular attention to ascarids. Ascarid eggs are extremely durable and resistant to adverse weather conditions. They can survive in the environment without herbage for up to 5 years. In general only young horses (or debilitated older animals) are at risk from ascarid infections as strong immunity develops with exposure and most animals self-cure by 18 months of age, although adults may harbor a few ascarids, which can act as source of infection for foals.
- While many anthelmintics (piperazine, benzimadazoles, pyrantels, ivermectin and moxidectin) have efficacy against ascarids it is generally shorter than their efficacy for other parasites and few if any are effective against migratory forms. Re-infection has been known to occur in as little as 2 weeks after treatment if the foal remains in a contaminated environment. In designing deworming programs for ascarids it is important to eliminate infection from mares to prevent environmental contamination and it is thus recommended to treat mares monthly in the last trimester of pregnancy.

Figure 4.84: Weanling with *Lawsonia intracellularis* infection. Note the poor body condition. Unlike most foals with the condition this foal did not have prominent submandibular edema.

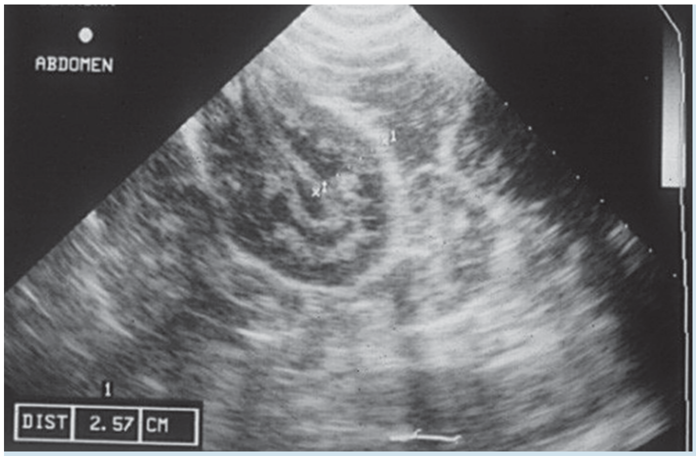


Figure 4.85: Ultrasonographic image of the abdomen demonstrating edema and thickening of the small intestine that is typically seen with *Lawsonia intracellularis* infection.

Lawsonia intracellularis infection (Figs 4.84 & 4.85)

History

Proliferative enteropathy caused by *L. intracellularis* typically affects 4–7-month-old foals. Multiple animals on the same farm may be affected.

Clinical signs

- Onset may be insidious and abnormal signs not identified until disease is advanced.

- Weakness, depression, fevers, ill-thrift, poor body condition and peripheral edema are most common.
- Diarrhea is not present in all cases and the hypoproteinemia is often more severe than would be expected considering the duration and severity of diarrhea.

Differential diagnosis

Other causes of hypoproteinemia and chronic weight loss include:
- infectious diarrhea of other etiologies
- malnutrition/poor diet
- inflammatory bowel disease (rare in this age group)
- intestinal neoplasia
- protein-losing nephropathy
- parasitism.

Diagnosis

- Total protein levels should be evaluated and monitored during treatment. Marked hypoproteinemia is almost invariably present.
- Serology can be useful.
- Detection of *L. intracellularis* in feces via PCR is diagnostic but a negative result does not rule it out.
- Testing for other enteropathogens should be performed because co-infection can occur.
- Thickened small intestine may be evident ultrasonographically. (Normal mural thickness <3.5 mm.)
- Surgical biopsy or necropsy may be required for a definitive diagnosis.

Treatment

- Macrolides can be used, although other antimicrobials such as chloramphenicol may also be effective.
- Tetracyclines (oxytetracycline, doxycycline) are the most commonly used antimicrobial in pigs with the same disorder and they have also been used successfully in foals.
- Supportive therapy, particularly oncotic support, is important, as is nutritional support. Parenteral nutrition is required in severely affected foals.

Rhodococcus equi infection

- Diarrhea as a result of *R. equi* infection is a common occurrence in endemic areas. In a study at the University of Kentucky diagnostic lab, 50% of *R. equi* pneumonic foals presented to necropsy also had intestinal manifestations characterized by granulomatous or suppurative inflammation of the Peyer's patches and the mesenteric and/or colonic lymph nodes. The majority of foals with *R. equi* pneumonia do not show clinical signs of the intestinal disease. It is also speculated that foals with subclinical intestinal manifestations may not gain body weight as readily as they should.
- Diarrhea rarely occurs alone. In the same study, only 4% of the foals with intestinal *R. equi* lesions did not have pneumonia. However, other symptoms such as bronchopneumonia, uveitis or joint infections may not be immediately apparent.
- Disease is most frequently seen between 2 weeks and 6 months. The peak age incidence varies with location and may vary with the local climate.
- In some cases bronchopneumonia is seen first and may be followed by diarrhea. In these cases it may not initially be clear if diarrhea is antibiotic-induced as a result of treatment for the bronchopneumonia or if it is related to the primary disease process.

Actinobacillus equuli infection

Actinobacillus equuli causes diarrhea in association with septicemia in foals less than 2 weeks old. There is a high mortality rate and identification of the organism on blood culture warrants aggressive treatment and a guarded prognosis. *A. equuli* septicemia is associated with profound depression and is often referred to as "sleepy foal disease."

Giardiasis

History

Giardia intestinalis, a protozoal parasite, is a recognized cause of enteric disease in some species but its role in foal diarrhea is currently unclear. Foals are probably exposed to *Giardia* through ingestion of feces of infected mares and other horses. Giardiasis, if it occurs, is probably more of a concern in immunocompromised foals.

Clinical signs

There is little known about the clinical presentation of giardiasis in foals. If it does occur, it presumably causes mild to moderate diarrhea, which is non-specific in character. Dehydration and anorexia could result in development of secondary clinical signs such as weakness.

Differential diagnosis

See pp. 101, 102.

Diagnosis

Diagnosis (and determination of the role of *Giardia* in foal diarrhea) is complicated by the fact that normal foals and horses can shed *Giardia*. Studies have reported shedding rates of up to 35% in normal foals. Therefore, detection of *Giardia* cysts in a diarrheic foal is not diagnostic. *Giardia* cysts or trophozoites can be visually identified in feces by direct evaluation of fecal smears or by concentration methods (i.e. zinc sulfate fecal flotation). Fluorescent antibody staining may also be used. Antigen tests (ELISA) are available but they have not been validated for use in horses and results should therefore be interpreted with caution.

Testing for other enteropathogens should be performed because of the unclear relevance of identification of *Giardia* and the potential for co-infection.

Treatment

- There has been minimal evaluation of treatment. Fenbendazole is used in other species, and its use in foals is reasonable.
- Metronidazole is considered to be less effective in other species but is still widely used and its use has been reported in an adult horse with presumptive giardiasis.
- Giardiasis is a zoonotic disease so appropriate precautions should be taken to prevent transmission.
- Nitazoxanide has been used in human medicine for the treatment of *Giardia*. In the United States, nitazoxanide is the active component in Navigator® an antiprotozoal medication used to treat equine protozoal meningitis. The dose that one of the editors (NMS) has used in foals is 25 mg/kg for 7 days.

Cryptosporidiosis

History

Cryptosporidiosis is caused by *Cryptosporidium parvum*, a protozoan parasite of many species. The overall role of *C. parvum* in foal diarrhea is unclear but there is reasonable evidence that it can be pathogenic. One study reported a significant association between the presence of *C. parvum* and diarrhea, and an increased incidence of fatal disease compared to other enteropathogens. However, subclinical infection is probably very common and disease may be more common in immunocompromised foals, particularly foals with severe combined immunodeficiency syndrome (SCID).

Clinical signs

Cryptosporidiosis is associated with mild to severe diarrhea. Signs of toxemia and systemic inflammation are not apparent initially but secondary effects including dehydration, depression and weakness can occur because of fluid and electrolyte loss, and anorexia. Death can occur. The prepatent period is 3–7 days.

Differential diagnosis

See pp. 101, 102.

Diagnosis

- *C. parvum* oocysts can be seen directly on fecal smears or with concentration techniques, but identification of them can be difficult. Oocysts are small and easily missed by examiners without ample experience.
- Acid-fast or other specific strains can be used to evaluate fecal smears and increase the sensitivity of diagnosis, as can direct immunofluoresence.
- Testing should be performed for other enteropathogens.

Treatment

- Cryptosporidiosis is a self-limiting disease. There are no known specific therapies for cryptosporidiosis in foals. Clinical signs usually persist for 5–14 days. During that time, supportive therapy, including fluid therapy and nutritional support, may be required.
- The prognosis should be good if appropriate supportive care is provided.
- This is a zoonotic pathogen so care should be taken to reduce the risk of human infection. Nitazoxanide has been used in human medicine for the treatment of *Cryptosporidium*. Its use in foals is reasonable (see Treatment of Giardiasis p. 112)

Coccidiosis

History

Eimeria leuckarti, E. uniungulsti and *E. solipedum* are coccidia that can be found in horses. *Eimeria leuckarti* is most commonly found and can be detected in up to 40% of healthy foals in some areas. The prepatent period of *E. leuckarti* is approximately 35 days. The role of *Eimeria* spp in foal diarrhea is unclear, and experimental infection of foals has not resulted in production of disease. As a result, if disease does occur, it is probably rare and perhaps more associated with immunocompromised animals

Clinical signs

If it exists, coccidiosis is presumably associated with mild to moderate diarrhea, without apparent toxemia.

Differential diagnosis

See pp. 101,102.

Diagnosis

Fecal flotation can be used to detect *Eimeria* oocysts. Testing for other enteropathogens should be performed.

Treatment

Treatment has not been described because *Eimeria* are not thought to be pathogenic in horses.

Colic (Figs 4.86 & 4.87)

Colic as an expression of abdominal pain in foals is extremely common. Abdominal pain in neonatal foals is always significant but the severity

Figures 4.86 & 4.87: Flank watching and rolling; typical signs of colic.

of the signs may not correspond to the severity of the lesion and thus the degree of pain is not a clear indicator as to the presence of a surgical lesion.

Diagnosing the exact cause of the abdominal pain may be challenging but a thorough examination is warranted as changes can occur quickly, resulting in a rapid deterioration of clinical condition.

Differential diagnosis of colic in foals by age group

0–7 days

- Congenital conditions
  - atresia ani/atresia coli (severe)
  - meconium impaction (moderate to severe)
  - lethal white (severe)
  - prematurity/dysmaturity (mild to moderate)
  - ruptured bladder syndrome (mild to moderate)
- Thoracic or abdominal trauma following rib fractures
- Infectious enteritis/colitis
- Gastric/duodenal ulceration
- Physiological ileus (electrolyte disturbances)
- Peritonitis
- Surgical lesions
  - complicated hernias – inguinal, scrotal or umbilical
  - intestinal volvulus or displacement
  - intussusception.

1–6 weeks

- Congenital conditions
  - ruptured bladder syndrome
- Thoracic or abdominal trauma following rib fractures
- Infectious enteritis/colitis
- Non-infectious diarrhea e.g. lactose intolerance, antibiotic-induced diarrhea
- Gastric/duodenal ulceration
- Duodenal stricture
- Strangulating obstructions
- Non-strangulating obstructions (Ascarid or feed-related impactions, intussusceptions, foreign bodies)
- Complicated hernias
- Other less common causes of colic:
  - adhesions (post surgery or following severe enteritis)
  - fecoliths in miniature horses
  - white muscle disease.

6 weeks to 6 months

As above and *Strongylus* spp related vascular thrombosis.

Impending enteritis / colitis (Figs 4.88 & 4.89)

History

Enteritis is a common problem in foals and can be caused by a variety of pathogens. Regardless of the cause, signs of abdominal pain may

Figure 4.88: Marked abdominal distension and colic in a foal with impending enteritis.

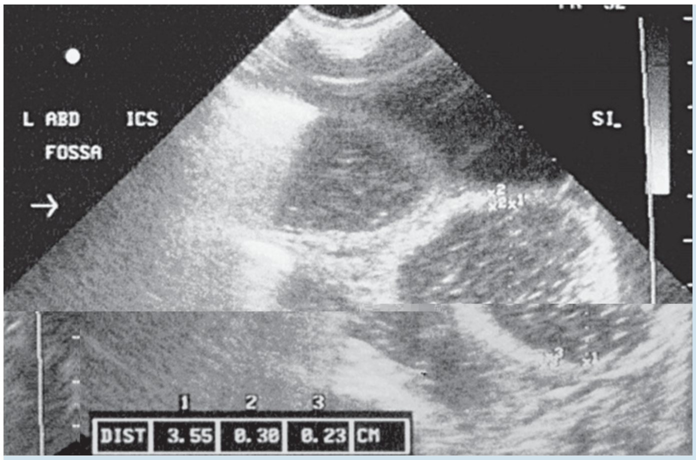


Figure 4.89: Ultrasonographic image of the small intestine demonstrating fluid-filled distended loops of small bowel in a foal with enteritis.

be present in diarrheic foals. Additionally, intestinal accidents (i.e. intussusceptions) can occasionally develop secondary to abnormal intestinal motility.

Clinical signs

Typical signs of abdominal pain are often observed in diarrheic foals and are usually apparent before the onset of diarrhea. These may range from mild and intermittent to severe, protracted and poorly responsive to analgesics. Some diarrheic foals will develop colic of such severity that exploratory surgery is required.

Differential diagnosis

Other possible causes of abdominal pain in diarrheic foals include:

- secondary intestinal rupture
- gastric ulceration
- gastric dilation.

Diagnosis

- A full examination should be performed on colicky diarrheic foals to ensure that a concurrent or secondary lesion is not present.
- Abdominal ultrasonography may demonstrate distended, hypermotile small intestine and/or fluidy, distended large intestine. Ultrasonographic examination is critical in severely colicky diarrheic foals. Poor intestinal motility and intestinal distension with progressive clinical signs are indicators that surgery should be considered.
- Gastroscopy can detect gastric ulcers.
- A nasogastric tube should be passed to rule out gastric reflux, which could be present from ileus, abnormal intestinal motility or secondary intestinal accident.

Treatment

- Supportive care and specific treatment as described for different causes of diarrhea is the most important aspect of treatment.
- Analgesics may be administered as needed. Excessive analgesia should be avoided so that progression of disease or development of a surgical lesion is not missed. Prokinetics may be required in some cases where colic signs are related to small intestinal ileus or gaseous distension of the large intestine (see p. 131 and Appendix 5).

Other conditions

Colon displacement

History

Large colon displacement occurs less often in foals compared to adult horses. Gaseous distension of the colon may predispose to displacement.

Clinical signs

- Characteristic signs of abdominal pain, including rolling, lying on the back, stretching, flank watching, tail flicking, stranguria and tenesmus may be present.
- Abdominal distension may be severe.
  - If colonic edema is noted along with unrelenting pain then an exploratory celiotomy will be warranted.
- Heart rate may be elevated consistent with the degree of pain. Signs of cardiovascular compromise or toxemia should not be present.

Differential diagnosis

See above.

Diagnosis

- Physical examination and monitoring of abdominal distension will help determine whether surgical intervention is required.
- Diagnostic imaging (ultrasound, radiography) may reveal large colon distension, but not a specific lesion.
- Definitive diagnosis is usually made at surgery.
- A nasogastric tube should be passed to detect nasogastric reflux, which may occur secondarily.

Treatment

- In some cases, displacements will correct spontaneously with supportive therapy. Pain control and fluid therapy are usually required. Temporary withholding of nursing may assist in decreasing intestinal distension and increase the likelihood of spontaneous correction.
- Surgery will be required in foals with severe, uncontrollable or progressive pain or abdominal distension. It should also be considered in foals that demonstrate colonic edema on ultrasonographic examination. If signs of toxemia or cardiovascular compromise develop, surgery should be performed immediately. At surgery, the colon can be decompressed and placed in its normal orientation.

Colon torsion (Figs 4.90–4.95)

History

Torsion of the large colon is uncommon in foals, but can occur. Vascular compromise can develop rapidly and large colon torsion is an emergency situation.

Clinical signs

- Progressive abdominal pain and distension are the most common signs.

Figure 4.90: Marked abdominal distension and colic in a foal with colon torsion.

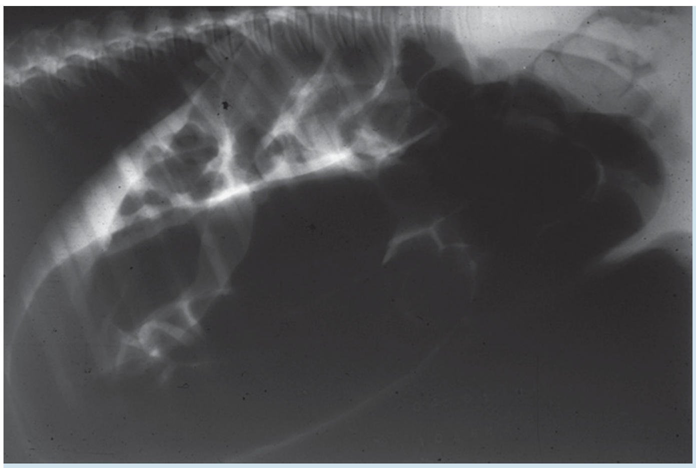


Figure 4.91: Abdominal radiograph from a foal with colon torsion demonstrating severe gaseous distension of the colon.

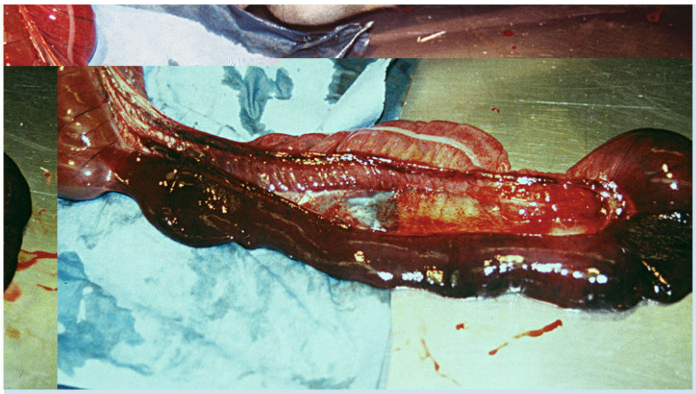


Figure 4.92: Colon torsion in a 1-day-old foal. Note the marked hyperemia associated with the compromised colon.

- Heart rate will be elevated consistent with the degree of pain and toxemia.
- Dehydration may be present, and can be severe.
- As vascular compromise develops, mucous membrane colour will deteriorate and other signs of cardiovascular compromise such as cool extremities and poor peripheral pulses may be observed. Affected foals can deteriorate rapidly, progressing from mild to intractable pain very quickly.

Differential diagnosis

Other causes of *abdominal distension* and *severe pain* include:

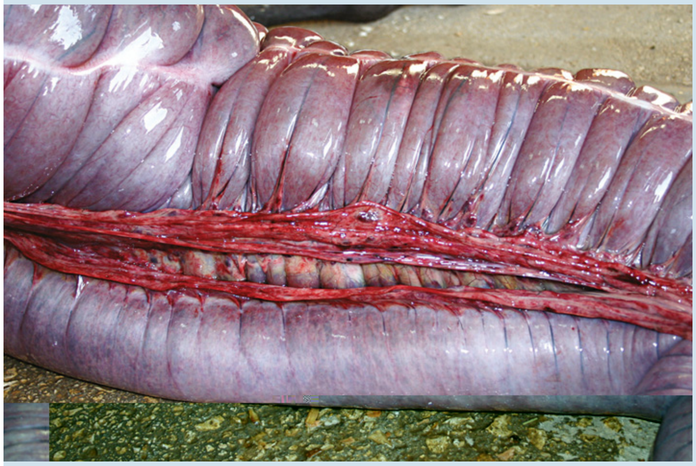
- enteritis (clostridiosis, salmonellosis)
- peritonitis, often secondary to gastric ulcer perforation
- other intestinal accident such as small intestinal volvulus
- meconium impaction
- atresia ani/coli
- lethal white foal syndrome.

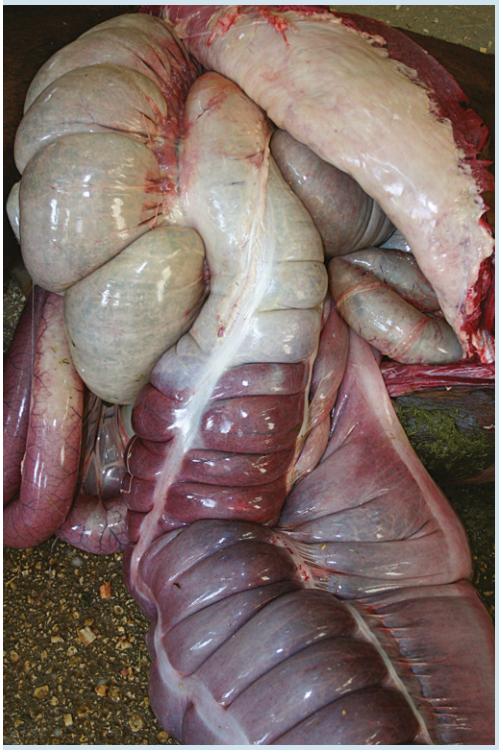
Diagnosis

- Definitive diagnosis is difficult without surgical exploration. The diagnostic plan should be aimed at determining whether surgical exploration is indicated.
- Abdominal ultrasonography and radiography will demonstrate intestinal distension and sometimes serosal edema primarily caused by large colon distension, but will not identify a specific lesion.
- Abdominocentesis may be useful to identify intestinal compromise. However, normal peritoneal fluid does not rule out large colon torsion.
- Blood gas analysis is useful to assess acid–base status, and guide preoperative therapy.
- In young foals (<2 days) the owners should be queried as to whether the foal has ever defecated, to rule out intestinal atresia.
- A nasogastric tube should be passed to check for reflux, which may occur secondarily.

Treatment

- Rapid surgical exploration is required. During preparation for surgery, intravenous fluid therapy should be started to maintain





Figures 4.93–4.95: Colon torsion in a 7-month-old weanling. Note the severe congestion of vessels and the demarcation between normal and compromised bowel.

perfusion, maintain blood pressure and attempt to correct any acid–base abnormalities.
- Analgesics will be required prior to surgery, either for transportation to a surgical facility or while preparing for surgery (see p. 131).
- Rarely, abdominal trocarization will be required to partially decompress the abdomen prior to induction of anesthesia. This should be reserved for situations where there is a delay in going to surgery or distension is so great that anesthetic complications are a serious concern. At surgery, the lesion can be identified and the viability of the colon assessed.

Small intestine obstruction (Figs 4.96–4.101)

History

Obstruction of the small intestine is uncommon in foals, and is typically associated with impaction of ascarids (*Parascaris equorum*). Ascarid impactions are most common in weanlings, particularly those with inadequate deworming programs. Foals with ascarid impactions typically develop clinical signs shortly after deworming, as adult worms are killed and lodge in the small intestine. Spontaneous ascarid impactions may also develop. Other possible causes of obstruction include impaction of intestinal contents (esp. ileal impaction) and foreign bodies.

Clinical signs

- Colic of variable intensity will be present. If gastric distension has developed, foals may be in severe pain.
- Abdominal distension may be present, but is typically less than with large colon disease.
- With simple obstruction, there should be no signs of toxemia or cardiovascular compromise because intestinal viability is not compromised.

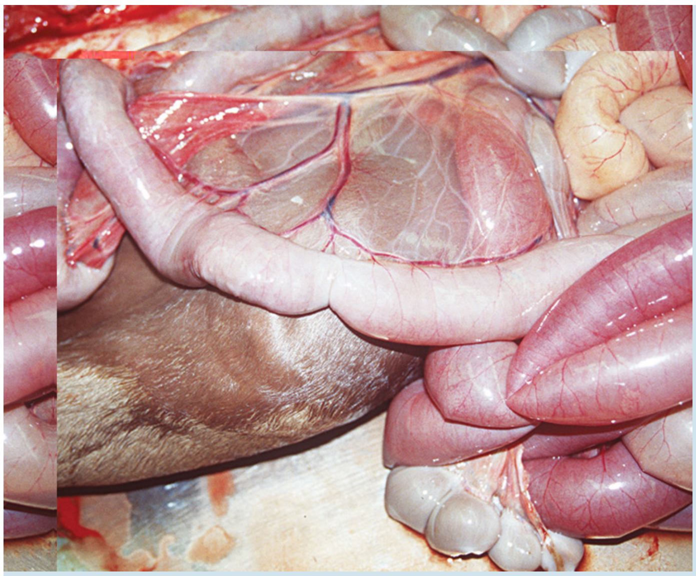


Figure 4.96: Jejunal impaction. Note the distended firm jejunum.

Differential diagnosis

Small intestinal distension can also be caused by:
- small intestinal strangulating lesion (i.e. volvulus)
- enteritis
- ileus.

Diagnosis

- Distended loops of small intestine are usually evident ultrasonographically. Occasionally, an obstruction (e.g. accumulation of ascarids) may be seen.
- Ascarid impaction should be considered likely in foals with an acute onset of severe colic with small intestinal distension that develops shortly after deworming. This is particularly true following late initial deworming.

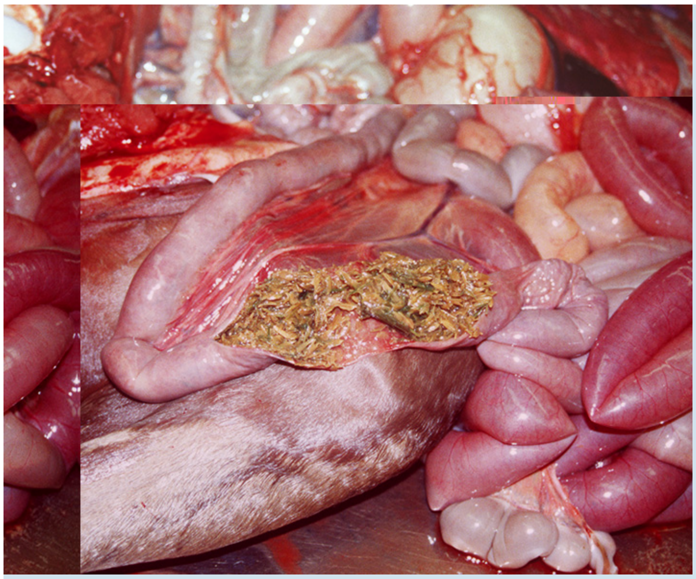


Figure 4.97: Same foal as in Fig 4.96. Note the impacted feed material within the jejunum.

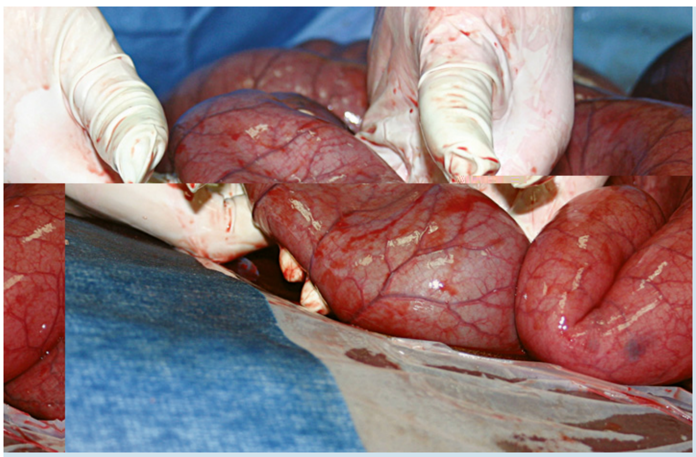


Figure 4.98: Ileal sand impaction. Note the impression that is left on the bowel following application of finger pressure.

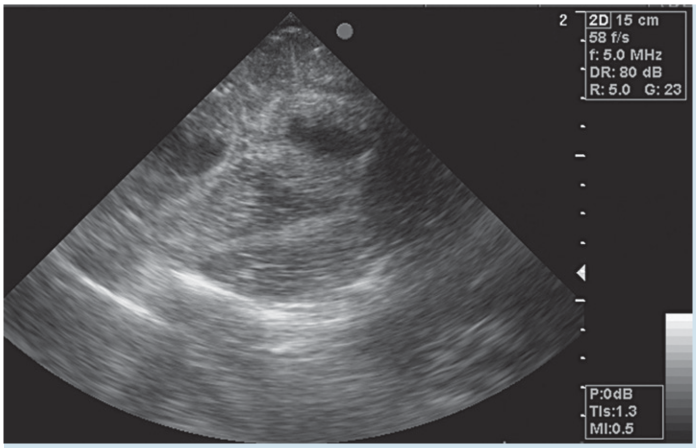


Figure 4.99: An ultrasonogram obtained from the ventral abdomen from a 16-day-old Thoroughbred foal that developed abdominal distension, nasogastric reflux and colic 12 days after an episode of diarrhea and fever which had initially responded well to antimicrobial and supportive therapy. There are several loops of distended small intestine, with varying wall thickness.

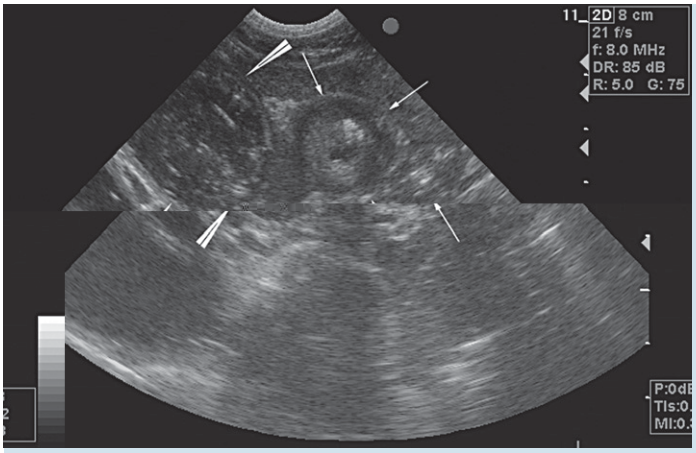


Figure 4.100: An ultrasonogram obtained from the ventral abdomen of the same foal as Fig 4.99. A portion of small intestine with an extremely thickened wall of varying echogenicity and a constricted lumen is visible (arrows). The adjacent loop of small intestine is distended but has a normal wall thickness (arrowheads).

Figure 4.101: A small intestinal mural abscess resected at celiotomy from the foal in Figs 4.99 & 4.100. Note the thickened wall and constricted lumen corresponding to the ultrasonographic findings illustrated in Fig 4.100.

- A nasogastric tube should be passed to determine whether gastric reflux is present. With large worm burdens, adult ascarids are sometimes present in gastric reflux.
- Abdominocentesis can be used to assess intestinal viability, which should be normal with simple obstruction.
- Exploratory surgery is often required for definitive diagnosis.

Treatment

- Withholding of feed and intravenous fluid therapy may result in resolution of feed-associated impactions and incomplete obstructions with ascarids. Osmotic cathartics should not be used.
- Frequent gastric decompression may be required.
- Often, surgery is required because of poor response to medical treatment, severity of clinical signs or concern that a strangulating lesion might be present. Surgery is usually required for ascarid impactions and foreign body obstruction. The prognosis for small intestinal obstructions requiring surgery is guarded.
- If ascarid impaction was present, deworming and pasture management should be addressed to prevent further cases on the farm.

Small intestinal volvulus (Figs 4.102–4.106)

History

Volvulus of the small intestine is one of the most common causes of surgical colic in foals, and is most common in 2–5-month-old foals.

Clinical signs

- Colic (rolling, flank watching, tail swishing, stretching) is the main initial sign and may be of sudden onset. Pain is progressive and can rapidly become severe, particularly as gastric distension develops.
- Poor response to analgesics is common.
- Signs of toxemia and cardiovascular compromise (prolonged CRT, hyperemic mucous membranes, cool extremities) may develop as the intestine becomes devitalized.

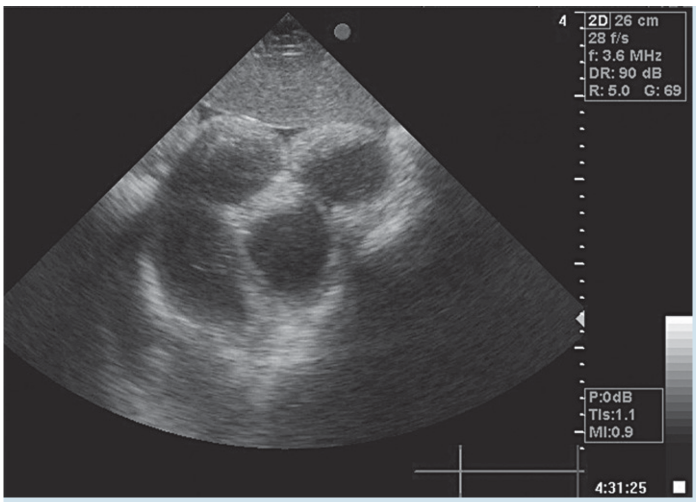


Figure 4.102: Ultrasonographic image from a yearling with small intestine volvulus. Note the distension of the SI and ventral sedimentation of intestinal contents.

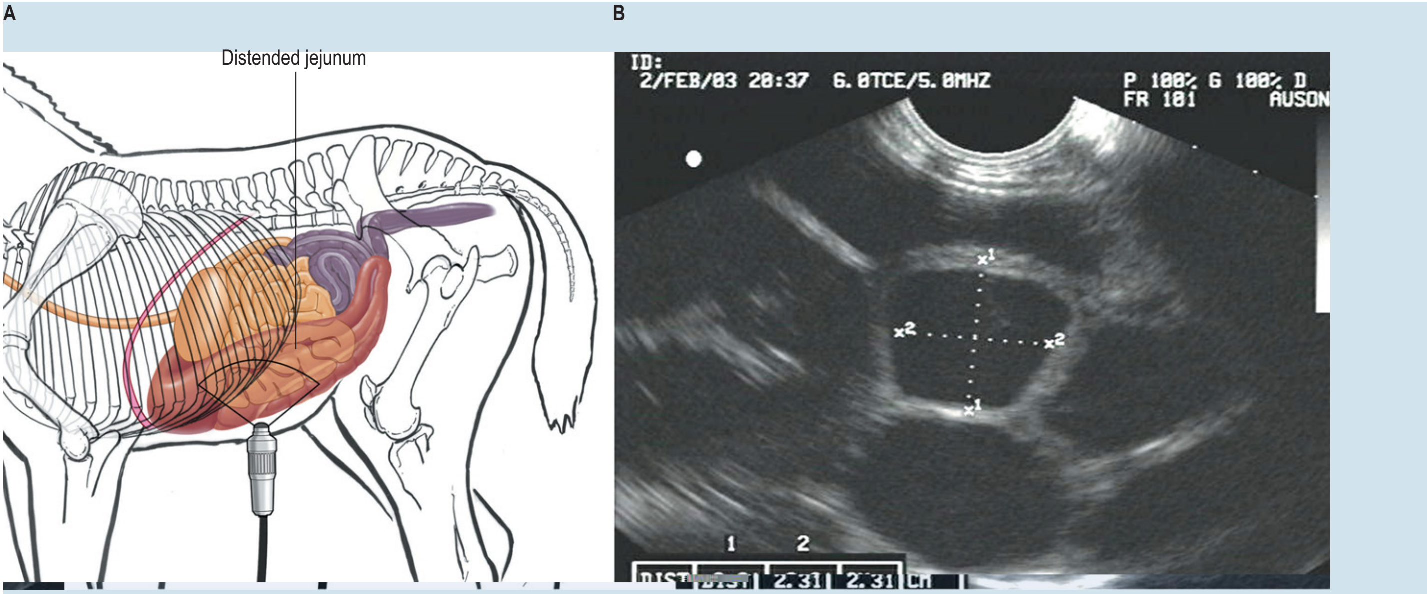


Figure 4.103: Schematic and ultrasonographic images of a foal's abdomen with a small intestinal volvulus. Note the marked small intestine distension (from Slovis NM 2003 Gastrointestinal failure. Clinical Techniques in Equine Practice 2:79–86).

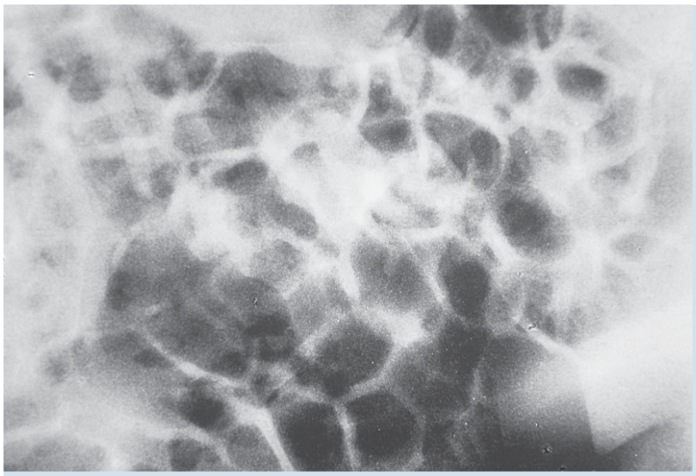


Figure 4.104: Abdominal radiograph from a foal showing marked gaseous distension of the small intestine. Exploratory celiotomy revealed a small intestinal volvulus.

Figure 4.105: Hyperemia of oral mucous membranes and a "toxic line" (marked hyperemia above the teeth) in a 10-day-old foal with a small intestinal volvulus.

Differential diagnosis

Other causes of small intestinal obstruction (see above).

Diagnosis

- Distended, hypomotile or amotile loops of small intestine are evident ultrasonographically with or without serosal edema and thickening (>3.5 mm).
- Nasogastric reflux may be present.
- Abdominal fluid may be normal, cloudy or hemorrhagic, depending on the degree of intestinal compromise.
- Definitive diagnosis is achieved at surgery.

Treatment

Early identification and surgical correction is required. The prognosis is better if the intestine appears viable after correction of the volvulus and no resection is required than if part of the small intestine must be removed. In some cases, there are no treatment options because of the amount of non-viable intestine.

Prognosis can be guarded because of the potential for postoperative complications, particularly adhesions.

Intussusception (Figs 4.107–4.114)

History

Intussusceptions are uncommon intestinal accidents that may develop spontaneously or secondary to disturbances in intestinal motility

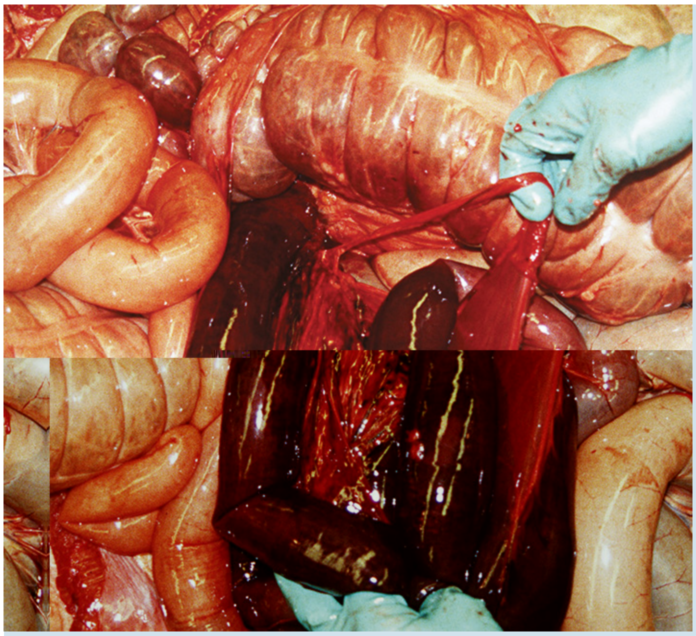


Figure 4.106: Small intestinal volvulus with entrapment in a mesenteric rent in a 7-month-old QH colt.

Figure 4.107: Gastric decompression in a 1-week-old foal with a jejuno-ileal intussusception.

(e.g. enteritis). Enteritis may be a predisposing factor. In older foals, tapeworm infestation may also be a risk factor.

Clinical signs

- Typically, intestinal obstruction causes development of acute and progressive signs of colic. Often, gastric decompression does not result in significant clinical improvement. Abdominal distension may occur but is not consistent.
- Signs of toxemia and cardiovascular compromise are uncommon initially but may develop with intestinal compromise or rupture.
- Sometimes, an incompletely obstructive intussusception may be associated with chronic colic.

Differential diagnosis

Other causes of colic without diarrhea include:
- small intestinal volvulus
- gastric/gastroduodenal ulceration
- impending diarrhea
- intestinal obstruction
- other intestinal accident.

Diagnosis

- A nasogastric tube should be passed. Nasogastric reflux will be seen with more proximal lesions such as jeuno-jejunal or jejuno-ileal intussusceptions but will develop over time with more distal lesions.
- Small intestinal distension will be evident ultrasonographically on the right side of the abdomen. Intestinal motility is variable, and the presence of distended but motile small intestine does not rule out intussusception. Often the intussusception may be detected ultrasonographically by imaging the characteristic "target lesion."

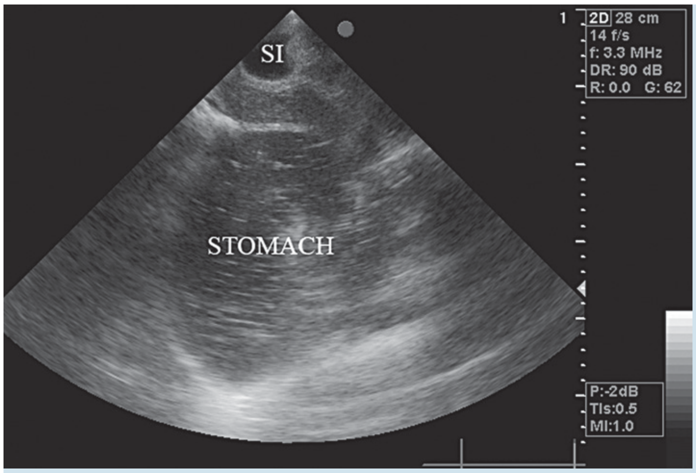


Figure 4.108: An ultrasonogram obtained from the cranio-ventral abdomen of a 24-day-old Thoroughbred foal that presented with colic due to a jejuno-jejunal intussusception. The stomach is markedly distended and a thick-walled loop of small intestine (SI) lies on the ventral floor of the abdomen.

- Abdominocentesis is useful to identify changes consistent with intestinal compromise (see p. 79).
- In the absence of an identified "target lesion," definitive diagnosis is made at surgery.

Treatment

Surgical correction is required in most cases. The decision to go to surgery should be based on the persistence of colic signs and reflux. On occasion intussusceptions may resolve spontaneously or following induction of general anesthesia. Prognosis depends on the amount of involved intestine and the type of surgery that was required, but is guarded to poor overall.

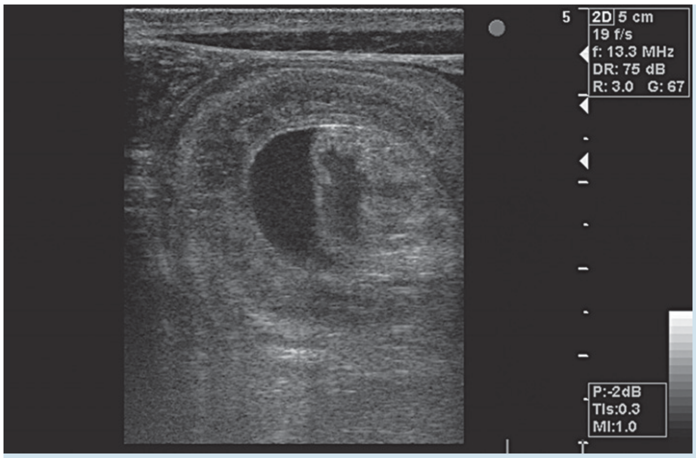


Figure 4.109: An ultrasonogram of the ventral abdomen of the foal in Fig 4.108. A thickened loop of intestine containing the intussuscepted portion lies on the ventral floor of the abdomen creating a "bulls-eye" appearance.

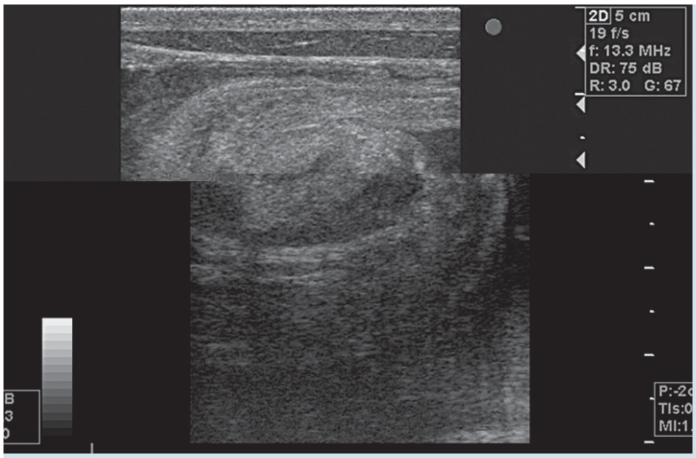
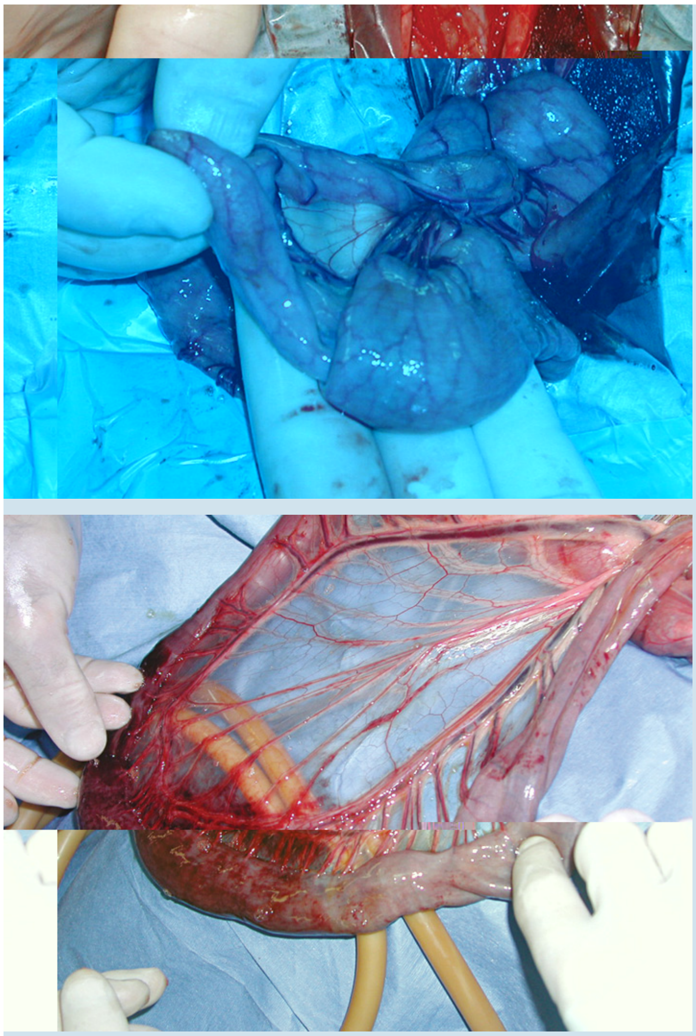


Figure 4.110: An ultrasonogram obtained from the ventral abdomen of a 24-day-old Thoroughbred foal that presented with colic due to a jejuno-jejunal instussusception. The portion of the jejunum that contains the intussusception is imaged in long-axis (same foal as Figs 4.108 & 4.109).



Figures 4.112 & 4.113: Prior to and following removal or reduction of an jejuno-jejunal intussusception. Note the vascular compromise of the vessels associated with the intussusceptum.

Figure 4.111: Findings at laparotomy of the foal in Figs 4.108 & 4.109. The surgeon is holding the intussuscepted portion of the jejunum.

Figure 4.114: Tapeworm on surgeon's glove. Ileo-cecal intussusceptions are classically associated with tapeworm infestations.

Hernias – umbilical, scrotal, inguinal (Figs 4.115–4.133)

History

Umbilical hernias are common congenital defects of the body wall with the incidence reported to be as high as 2% in Thoroughbred foals. Body wall hernias other than umbilical hernias can occur following surgery or trauma.

Inguinal/scrotal hernias are also seen in young colts and can be present at birth or may develop within several days and are related to large inguinal rings, trauma, and increased abdominal pressure and straining. Inguinal hernias are usually unilateral but can be bilateral and are commonly seen in Tennessee walkers and Standardbred.

Most congenital inguinal hernias are indirect, with the intestines passing through an intact vaginal ring and contained within the parietal layer of the vaginal tunic. Indirect hernias are usually reducible, are not life threatening and usually resolve with manual reduction within a few days. A direct hernia occurs when the parietal vaginal tunic or the peritoneum in the vaginal ring region tears and the intestines become positioned under the skin. Direct hernias are normally irreducible, contain large amounts of intestine, are life threatening and regarded as a surgical emergency.

An uncomplicated hernia is any type of hernia which either does not contain intestine or contains non-incarcerated intestine. A complicated hernia is any type of hernia which contains incarcerated or strangulated intestine.

Clinical signs

- Hernias are usually apparent visually or detected by palpation. In most cases of umbilical or inguinal hernia, there are no clinical abnormalities.
- Individual loops of small intestine may be apparent under the skin in foals with direct inguinal hernias. The inguinal skin is thin and

Figure 4.115: Large uncomplicated umbilical hernia.

Figure 4.116: Complicated umbilical hernia in a 2-month-old foal. The hernia was noted to have increased in size in association with mild colic signs. The hernia was palpably firm and irreducible.

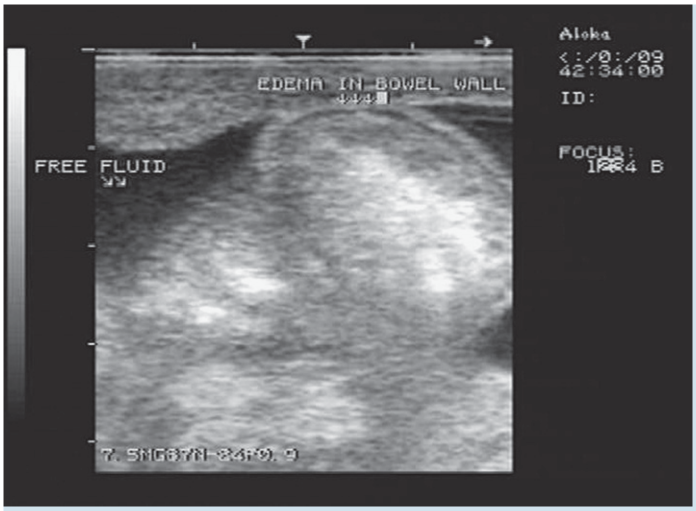


Figure 4.117: Ultrasonographic image of the umbilical region of the foal in Fig 4.116. Note the incarcerated intestine that has become thickened and edematous.

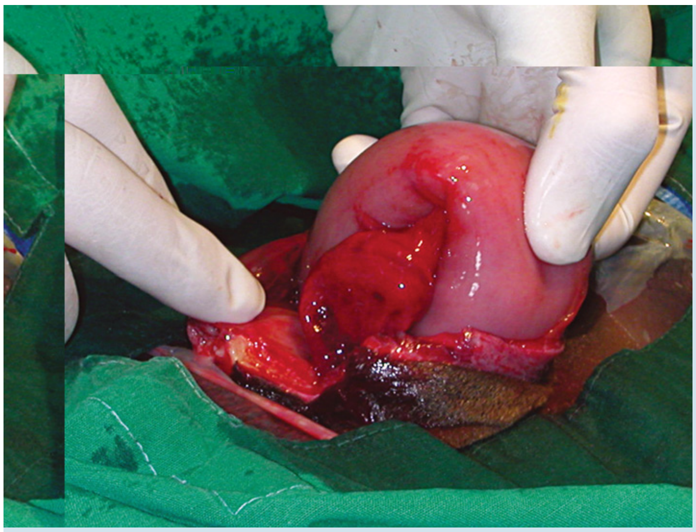
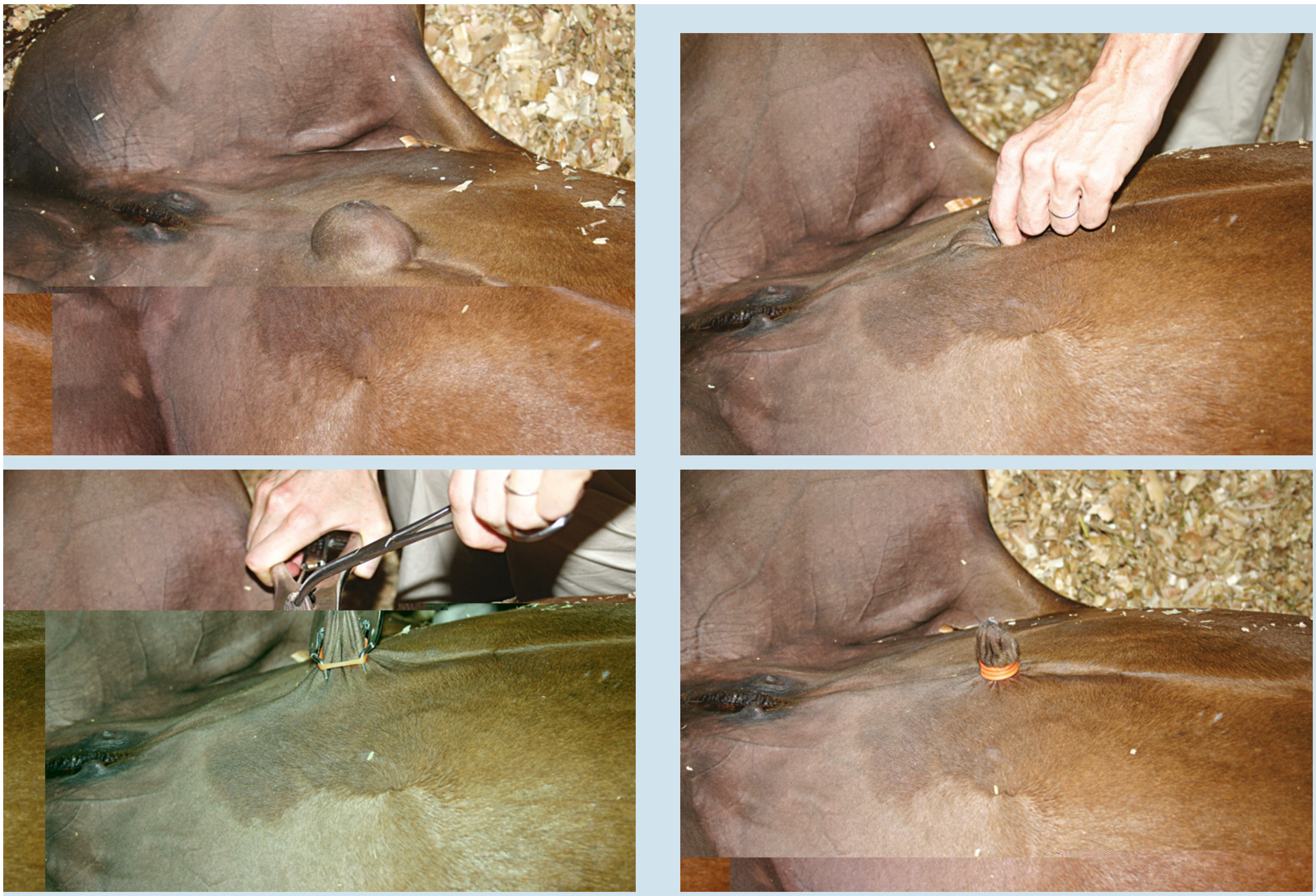


Figure 4.118: Same foal as Figs 4.116 & 4.117 during surgery. Note the congestion of the vessels associated with the section of ileum that had become incarcerated.



Figures 4.119–4.122: Application of rubber rings to an umbilical hernia. Placing the foal on it's back following sedation helps to avoid entrapment of intestine within the rings. Following reduction of the hernia, the skin is grasped with forceps and 3–4 rings are placed as close to the body wall as possible.

Figure 4.123: Umbilical hernia 1 week after the application of rubber rings. Localized swelling around the umbilicus is normal.

associated stretching may result in tearing of the skin or disruption of dermal circulation.

- Signs of colic are often but not always observed when strangulation has occurred, but will develop as intestinal compromise progresses and intestinal distension develops.

Differential diagnosis

Other similarly appearing masses include:

- abscess
- hematoma
- seroma.

Diagnosis

- Physical examination, including palpation of the hernia, is typically diagnostic.
- The hernia should be reduced, if possible, and the ease of reduction noted. The presence of intestine within the hernia is not necessarily a significant concern unless it cannot be reduced.
- Ultrasonographic examination can differentiate non-reducible hernia from an abscess, and characterize the hernia contents.

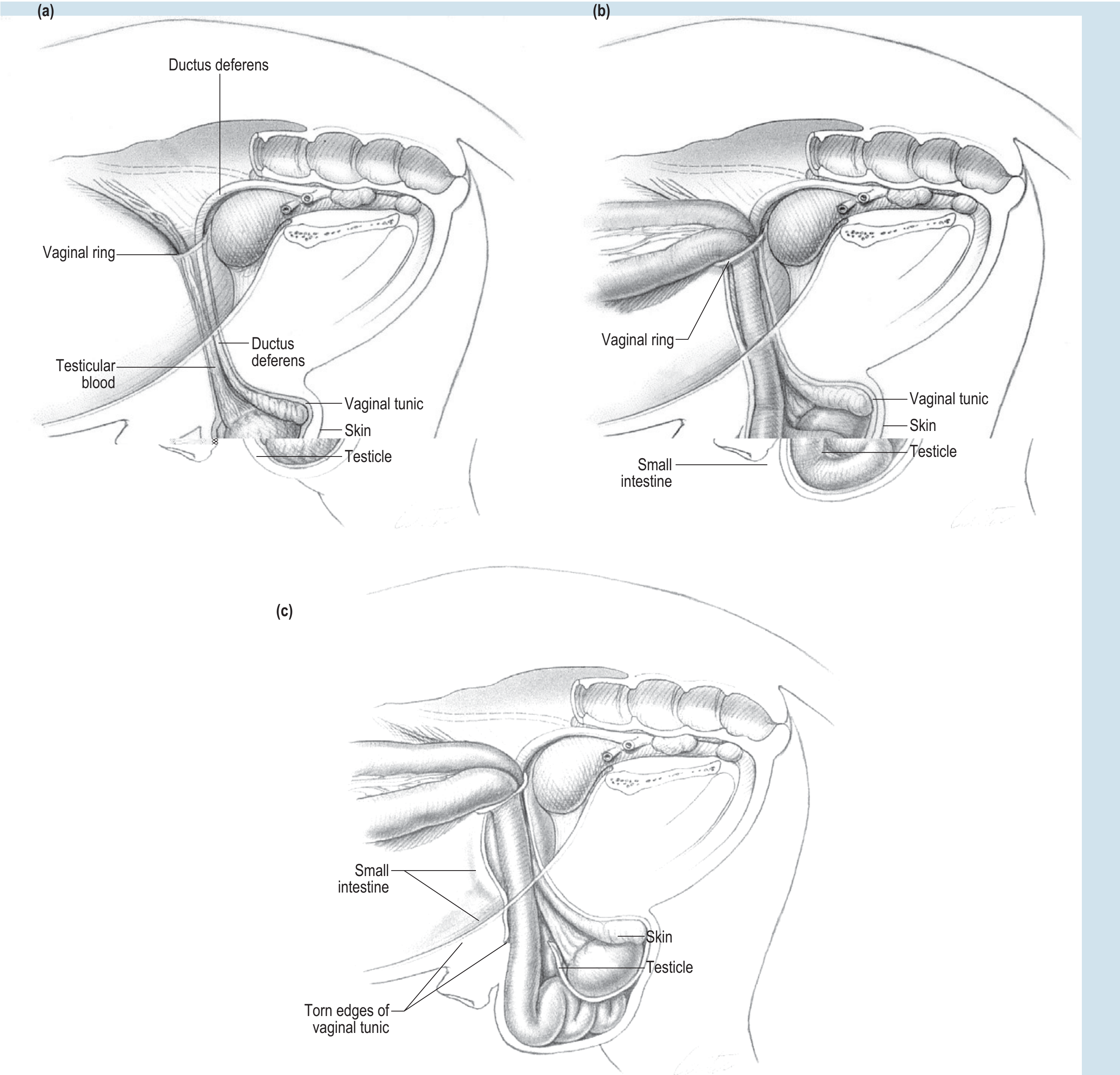


Figure 4.124: (a) Normal inguinal structures. (b) Indirect inguinal hernia. (c) Direct inguinal hernia. The difference between b and c is that the tunic has ruptured to allow the bowel to escape into a subcutaneous position. (From Bartmann C et al 2002 Diagnosis and surgical management of colic in the foal: literature review and retrospective study. Clinical Techniques in Equine Practice 1:125–142).

- Signs that suggest strangulation include an inability to reduce the hernia, increased hernia size, pain, heat, increased firmness, edema and pain on palpation. Strangulation should be suspected in any foal with a hernia that develops colic.

Treatment

- A conservative approach is usually indicated initially. Small hernias usually correct without treatment in the first few weeks of life. Owners should be instructed to monitor the size of the hernia and reduce it frequently. Any change in clinical condition of a foal with a hernia, particularly a large hernia, should result in prompt veterinary examination. Bandages or hernia clamps can be used to maintain reduction and allow for spontaneous closure of the hernia ring. Hernia clamps/rubber rings may facilitate closure by stimulating scar tissue formation. Pressure sores are a potential complication of bandaging for inguinal hernias.

Figure 4.125: Indirect inguinal hernia in a 4-month-old colt.

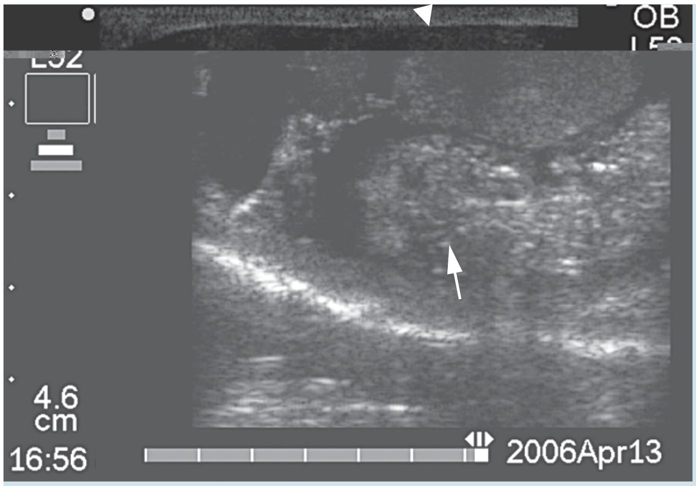


Figure 4.126: Ultrasonographic image of the scrotum from the same foal as Fig 4.125; note the intestine (arrow) beside the testicle (arrowhead).

Figures 4.127 & 4.128: Bandaging to facilitate inguinal/scrotal hernia reduction.

Figure 4.129: Direct inguinal/scrotal hernia. Note the intestine situated under the skin and the tearing of the scrotal skin. This foal required cardio-respiratory resuscitation on presentation and surgery was not performed as he was considered too great an anesthetic risk. This hernia was manual reduced and an inguinal bandage placed which was changed daily for two weeks. The hernia did not recur and the colt was castrated by a closed technique as a weanling.

Figure 4.130: Direct inguinal/scrotal hernia in which strangulation of the small intestine has occurred.

Figure 4.131: Large body wall hernia in a 4 month-old foal, following a splenectomy at 45 days of age.

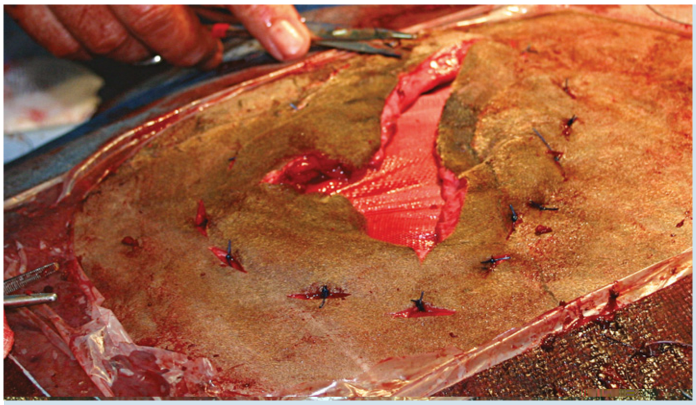


Figure 4.132: Mesh repair of the hernia in Fig 4.131.

Figure 4.133: The foal in Fig 4.131 seen here as a yearling.

- Larger hernias, for example umbilical hernias greater than 5 cm in diameter, are more prone to complications and less likely to correct spontaneously.
- In general, elective surgical correction is indicated for large hernias (i.e. umbilical hernia >7–10 cm) or ones that have not resolved by 7–9 months of age.
- If a strangulating hernia is suspected, prompt surgical intervention is required. Concurrent removal of the testicle is recommended in scrotal or inguinal hernias to facilitate closure of the inguinal ring.

Other causes of colic

History

A variety of other causes of colic can occur, including displacements, impactions, incarcerations, intussusceptions, ileus and obstructions other than those described above. Foals that are unable to nurse normally may be more likely to develop intestinal obstructions associated with inappropriate ingestion of feed and non-feed items. Miniature horse foals are prone to development of fecoliths.

Clinical signs

Characteristic signs of abdominal pain include rolling, lying on the back, flank watching, tail flicking, stranguria and tenesmus. Partial or complete anorexia is common. Abdominal distension may be present. Heart rate is usually elevated consistent with the apparent degree of pain. Pyrexia is uncommon.

Differential diagnosis

Signs of abdominal pain are non-specific and can be caused by a variety of other intestinal diseases including:
- enteritis
- lactase deficiency.

Non-intestinal diseases that may appear similar include:
- ruptured bladder
- peritonitis.

Diagnosis

- A full physical examination is required to differentiate colic from similarly appearing non-intestinal diseases.
- A nasogastric tube should be passed to detect gastric reflux.
- Diagnostic imaging (ultrasound, radiographs) can be useful to identify a specific lesion or assist in deciding whether surgery is indicated.
- Gastroscopy is useful, particularly in mild or chronic cases, to rule out gastric ulceration.
- Abdominocentesis can help assess bowel viability and assist in ruling out other conditions such as peritonitis and bladder rupture.
- Serial measurement of abdominal distension can be used to monitor progression of disease.

Treatment

- Treatment depends on the specific cause.
- Analgesics are often required (see p. 131).

- Intravenous fluid therapy may be required in foals that are dehydrated or anorexic. Nutritional support may also be required.
- If nasogastric reflux is present, frequent gastric decompression is critical. If intestinal or gastric distension is present, restriction of nursing may be required.
- In mild/moderate cases, empirical treatment for gastric ulceration can be attempted.
- Surgical intervention may be required in some cases.

Abdominal abscesses

History

Infection of the umbilical vein, umbilical arteries or urachus is a common cause of abdominal abscessation. There may be a history of prior umbilical disease.

Localized peritonitis from a small duodenal ulcer may produce an abscess, and a history of clinical signs consistent with gastroduodenal ulceration may be present; however diffuse peritonitis is more common following intestinal perforation.

Rhodococcus equi abdominal abscesses have been identified in older foals.

Abscessation of abdominal lymph nodes can occur following any bacteremic event. Abscesses may develop following abdominal surgery, particularly if an enterotomy or resection has been performed. Sometimes, there is no history of a preceding event that would predispose to development of an abdominal abscess.

Clinical signs

The clinical presentation can be highly variable. Intermittent fever, depression, weakness and poor body condition may be the only apparent signs. If the abscess is affecting passage of ingesta, or has resulted in intestinal adhesions, colic may occur.

Abdominal abscesses are occasionally identified incidentally during colic surgery. Small abscesses that are not associated with important structures or affecting intestinal motility may be present without any obvious clinical signs. Sometimes, these occult abdominal abscesses will rupture and the first signs noted will be that of acute septic peritonitis. These include fever, weakness, depression, cardiovascular shock, signs of systemic toxemia, abdominal distension, abdominal pain or sudden death.

Differential diagnosis

- Intermittent fever, depression and poor body condition can also be caused by:
  - lung abscesses
  - enteritis
  - *Lawsonia intracellularis* infection
  - neoplasia.
- Other causes of septic peritonitis include:
  - perforated gastric ulcer
  - perforated duodenal ulcer
  - rupture of intestinal viscera.

Diagnosis

- Careful palpation of the external umbilicus should be performed; however a normal external umbilicus does not rule out abscessation of internal umbilical structures.
- Leukocytosis consisting mainly of a neutrophilia will almost always be present, as will a moderate to marked increase in plasma fibrinogen.
- Blood culture may identify the etiologic agent if bacteria are periodically being shed from the abscess. Blood culture is best performed prior to initiation of antimicrobial treatment and during an episode of pyrexia.
- An echogenic mass consistent with an abscess may be evident via ultrasound (see Fig 10.35 p. 289).
- Abdominocentesis should be performed, ideally after ultrasonographic examination so that a fluid pocket might be identified and an abscess is not inadvertently tapped.
- Aspiration of an abscess identified ultrasonographically should only be performed if there is clear evidence that the abscess has a distinct wall that is fused to the body wall. Otherwise, aspiration could result in contamination of the abdomen with subsequent septic peritonitis.
- Thoracic radiographs/ultrasonography are indicated to rule out concurrent lung abscesses if *R. equi* is considered a possible cause.
- While currently of limited availability, advanced imaging techniques such as CT and MRI can be useful in small foals.
- Exploratory celiotomy is often required for definitive diagnosis.

Treatment

- Long-term antimicrobial therapy may be effective in some cases with small abscesses and no active, underlying disease. Therapy is ideally based on culture and sensitivity data; however empirical selection is usually required. Erythromycin, azithromycin or clarithromycin with or without rifampin are reasonably effective for abscesses caused by Gram-positive organisms.
- Surgical resection is rarely possible. If an abscess is associated with a discrete intestinal loop, resection of the abscess and intestine may be possible. Rupture of an abscess during surgical manipulation is a concern. Marsupialization of the abscess may be possible in some cases.
- Hyperbaric medical therapy has been used in a limited number of foals with internal abscessation. Outcome for foals treated with hyperbaric medicine and diagnosed with an abscess <9 cm in size has been promising. More research is needed with regard to this treatment modality and it may be cost prohibitive in many cases.
- Overall, the prognosis is guarded to poor.

Miscellaneous

Feeding the orphan foal (Figs 4.134–4.138)

Orphan foals are not uncommonly encountered, and early measures are required to maintain their health status, particularly in very young foals.

Colostrum feeding

In foals that are orphaned shortly after birth, it is essential to ensure that adequate transfer of maternal antibodies is achieved. In the first 12 hours of life, colostrum (fresh or frozen) or plasma can be fed. After

Figure 4.134: Orphan foal feeding from bucket.

Figure 4.135: Foal drinking from a calf feeding bucket with teat.

Figure 4.136: Orphan foal nursing from a goat.

Figure 4.137: Orphan foals being kept together for company.

this time, plasma must be administered intravenously. Immunoglobulin G levels should be checked approximately 12 hours after feeding or 24 hours after plasma transfusion to ensure that adequate levels have been provided. (See Chapter 11, p. 298).

Nurse / foster mare

The best means of feeding an orphan is a nurse mare. This is the preferred approach because it provides a normal diet and also aids in normal social development. Most foals will readily accept a nurse mare. The mare may be slower to accept the foal and many techniques such as blindfolding, restraints and sedation may be used to aid in acceptance of the foal.

With the increased value of Thoroughbred mares and foals, it is now increasingly uncommon for mares that have recently lost a foal to be used as foster mares. Most mares are supplied from agencies and are commonly mixed breed mares that foster different foals every year. It is important to choose a reputable agency that supplies mares in good health with up-to-date vaccinations. The advantages are that such mares will frequently accept a new foal with minimal fuss and assistance may be given with the fostering process.

Hand rearing

If a nurse mare is not available, milk replacer will be required. There are many different brands available and the manufacturer's instructions should be followed with respect to quantity of feed and correct mixing technique. The use of an inappropriate milk replacer or improper dilution can result in diarrhea and other problems.

Underfeeding is a common problem in hand-raised foals. Foals drink approximately 20–25% of their body weight (BW) each day once they reach a week of age. Initial feeding programs should start at 10% of BW and increase by 1–2% until the goal of 20–25% is reached. Therefore, a 60 kg foal requires approximately 12–15 liters

Figure 4.138: Orphan foal with old pony as company.

Table 4.1: Suggested feeding plan for Thoroughbred foal with expected mature bodyweight of 500 kg

AGE (weeks)	WEIGHT (kg)	FEEDS/DAY	AMOUNT OF MILK/FEED	CONCENTRATE FEED/DAY (g)
1	50–60	12 (every 2 h)	500–750 mL	None
2	60–70	12 (every 2 h)	850 mL–1.0 L	0–100
3	70–80	12 (every 2 h)	1.1–1.4 L	100–200
4	80–90	9 (every 2 h	1.8–2.0 L	200–350
5	90–100	with 6 h break	2.0–2.2 L	350–450
6	100–107	overnight)	1.9–2.0 L	450–550
7	107–115		1.7–1.8 L	550–650
8	115–123		1.6–1.7 L	650–800
9	123–130		1.6 L	800–950
10	130–137		1.6 L	950–1150
11	137–143		1.5 L	1150–1350
12	143–150		1.5 L	1350–1550

The suggested feeding rates in the table above are a guide only and should be adjusted according to body weight and condition of the foal. The first day of life the foal should be fed at a minimum 10% of its body weight and *slowly* increased by 1–2% a day until 20% is reached (i.e. 50 kg foal fed 10% of body weight equals 5000 mL of milk to be consumed over 24 hours). Regular weighing and body condition scoring are essential. Foals should gain approximately 1.3–1.5 kg/day during the first 12 weeks – if satisfactory weight gains are not achieved, veterinary advice should be sought.

of milk replacer daily. See Table 4.1 for estimation of volume requirements.

Feedings should be at least every 1–2 hours over the first 24–48 hours and the interval between feeds can then be gradually increased along with the volume given per feed.

Should commercial forms of milk replacers not be available there are a number of other options:

- Goat milk is well tolerated by foals and causes few problems (increase in firmness of stools and metabolic acidosis have been seen). No alterations are usually made but it is generally difficult to obtain and may be expensive.
- Make your own: 250 mL of 2% cow's milk with 1 tsp of dextrose or white corn syrup added. This works well but again may be cost prohibitive. It may provide a "stop gap" remedy until commercial forms of milk replacer can be obtained.
- Human and calf milk replacer are not suitable.

Close attention should be paid to the foal's body condition and weight. Daily weight gains of 1.3–1.5 kg should be attained in *Thoroughbred* foals.

Bucket vs bottle

Buckets are one way to provide milk replacer. Foals can usually be trained to drink from a bucket with few problems. An advantage of bucket feeding over bottle-feeding is that the foal has more control over timing and volume of intake.

Bottle-feeding is another option; however it is the least desirable. Bottle-feeding is time consuming and may result in inappropriate bonding of the foal with humans, which could result in behavioral problems later in life. Bottle-feeding is best reserved as a temporary measure while a nurse mare is being obtained.

Other feedstuffs and socialization

Foal replacer pellets/foal creep feed should be offered from one week of age. Foals should have access to hay because normal foals will ingest small amounts of their dams' diet.

Company, ideally a quiet older horse, is important for proper social development and development of correct eating habits. Orphan foals that are reared on their own may be slow to accept feedstuffs other than milk. This may not be a problem in the early stages but as the foal gets older it will make it difficult to attain adequate weight gain.

Goats or sheep can be used as company if a suitable horse is not available. Under no circumstances should an orphan be placed with other mares and foals. Many mares will show very aggressive tendencies towards any foal that is not their own.

While a foal should be able to handle the transition to a solid diet at 2–3 months of age, if required, it is best not to wean foals off milk replacer until they are 3–4 months of age.

Feeding the sick foal

Nutritional support is often required because feeding is being restricted or withheld, or the foal is unable or unwilling to ingest an adequate volume of milk.

Enteral nutrition

Feeding milk or milk replacer via an indwelling nasogastric tube can be very useful because it allows for feeding of a normal diet. Flexible tubes are preferred and they should be securely fastened. Potential complications include aspiration pneumonia and laryngeal/pharyngeal irritation.

Feeding should be introduced gradually, start at 5% of BW and increase by 1–2% every 12 hours if feeding is tolerated. Sick foals should be fed regularly (hourly if possible) so that large volumes per feeding are not required. The nasogastric tube should be checked before every feeding, and replaced if there is any concern that it may no longer be in the proper location. Foals should be fed standing or in sternal recumbency, and kept upright for a period of time after feeding to reduce the ever-present risk of aspiration. It is wise to check for gastric reflux before each feeding. Mare's milk is preferred, although foal milk replacers are also useful. Water or glucose containing electrolyte solutions cannot provide adequate energy intake, although they can be useful in combination with parenteral nutrition to nourish enterocytes in foals unable to tolerate milk.

Parenteral nutrition (Figs 4.139 & 4.140)

In some cases, enteral feeding cannot be tolerated due to gas production resulting in abdominal distention and pain. If enteral feeding results in signs of colic it should be decreased to a tolerated level or stopped. This is most commonly seen in foals demonstrating signs of unreadiness for birth, septicemic foals and foals with severe enterocolitis. If enteral feeding cannot meet the foal's energy

Figure 4.139: Commercial TPN.

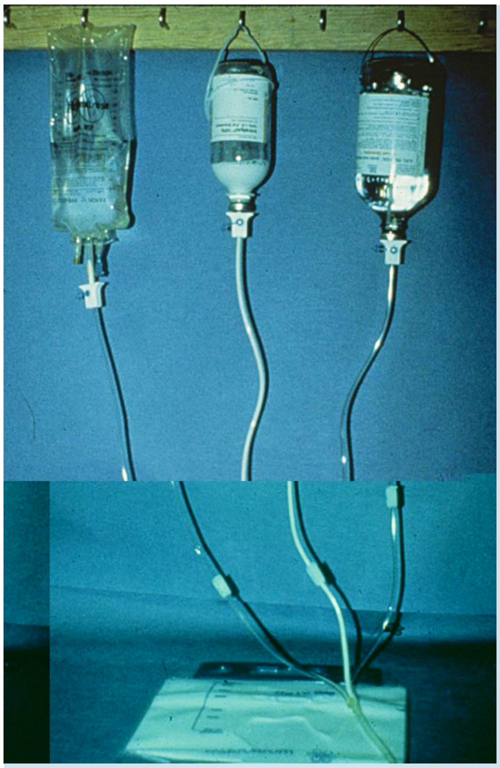


Figure 4.140: "Home-made" TPN.

requirements, total (TPN) or partial parenteral nutrition (PPN) is required. Parenteral nutrition is often avoided due to a perceived difficulty of administration and expense. However, practitioners who use it frequently would testify to its ease of use and overall economic benefits as a result of shortened hospitalization periods.

Ideally parenteral feeding should be delivered through a separate intravenous (jugular) catheter, or a double- or triple- lumen catheter (prevents contamination), but may be delivered through a single-lumen catheter.

Maintenance energy requirements should be calculated, and reached over the first 2–3 days. Calculating energy requirements is not always straightforward as there are no values available specifically for neonatal foals. In addition the extra nutritional requirements of sick foals are unknown. The total energy requirement (TER) of a sick 50 kg foal is estimated to be 50–70 kcal/kg/day. One protocol for administration of PN is to start at 33% of TER for 24–48 hours, followed by an increase of 33% over a further 48 hours provided blood glucose measurements are within normal levels. In most cases only partial parenteral nutrition is required as many foals have recovered sufficiently in a 3–5 day period to commence partial or complete enteral nutrition. Solutions for parenteral nutrition may be purchased commercially and various formulations are available. Two such commercial products are in Tables 4.2 and 4.3 and consist of:

Baxter ^{R} solution for parenteral nutrition (2 L)

- 400 mL 20% lipid (provides 950 kcal)
- 800 mL 10% amino acids (provides 256 kcal)
- 800 mL 40% glucose (provides 1280 kcal)

Given the energy requirements (50–70 kcal/kg/day) of a 50 kg foal the following amounts of this solution would be required for partial and total parenteral nutrition:

Table 4.2: Administration rates for Baxter^{R} solution for parenteral nutrition (2 L)

% TER	mL/day	mL/h
33%	660–800	28–33
66%	1320–1600	55–67
100%	2000–2400	83–100

Braun[R] solution for parenteral nutrition (2.5 L)

- 500 mL 20% lipid (provides 950 kcal)
- 1000 mL 8% amino acids (provides 320 kcal)
- 1000 mL 16% glucose (provides 640 kcal)

Given the energy requirements (50–70 kcal/kg/day) of a 50 kg foal the following amounts of this solution would be required for partial and total parenteral nutrition:

Table 4.3: Administration rates for Braun[R] solution for parenteral nutrition (2.5 L)

% TER	mL/day	mL/h
33%	1000–1300	42–54
66%	2100–2500	87.5–104
100%	3200–3800	133–158

A “home-made” solution for partial parenteral nutrition can be compounded from 50% dextrose (1000 mL), 20% lipid (500 mL), and 10% amino acids (2000 mL). This solution is ideal for use in foals with enterocolitis. Dextrose is the cheapest component but providing all of the required calories with dextrose alone would result in an extremely hyperosmolar solution; the administration of which would likely result in phlebitis and glucose intolerance. It is important to remember that foals with severe protein-losing enteropathy are at increased risk for thrombophlebitis due to loss of antithrombin III and the administration of hyperosmolar solutions would increase the risk for developing phlebitis.

Strict aseptic technique is required during preparation. Ideally laminar flow hoods should be used but if not available careful aseptic mixing may suffice. Dextrose and amino acids are mixed first, followed by the lipid to minimize lipid demulsification. A batch is split in half and divided between two separate sterile fluid bags. The first bag is used immediately and the second can be refrigerated for 24 hours.

Based on the calorific content of the above compounded solution the following rates of administration would apply:

Table 4.4: Administration rates for “home-made” solution for parenteral nutrition.

% TER	mL/day	mL/h
33%	1200	50
66%	2400	100
100%	3600	150

When choosing whether to use a commercial product or determine a mix for a “home-made” solution it is important to bear in mind the disease process involved in the affected foal. Sepsis for example results in glucose intolerance and limits glucose infusion rates. Premature foals have increased demand for protein. These considerations should be taken into account not only for determining the compounding ingredients but also in the timing of monitoring tests.

Blood glucose measurements should be performed every 4–6 hours and be within a range of 80–180 mg/dL. If values fall outside this range adjustments in the rate of crystalloid administration or percentage of dextrose content can be made. If a commercial solution is being used or if for some other reason it is not possible to vary the dextrose content adjustments of 10% TER (i.e. 15 mL/h for a 50 kg foal for the above “home-made” solution) should be made.

Measurement of serum triglycerides and ammonia is especially important in foals unready for birth and should be checked daily for the first 3 days to assess tolerance of the amino acid and lipid component. Values outside normal ranges (see Appendix 3) warrant adjustments in the components of the solution, or alteration of flow rates and more frequent assessments.

Parenteral nutrition should not be withdrawn quickly; rather it should be tapered over 1–2 days as enteral feeding is started.

Analgesic therapy for colicky foals

Analgesic therapy is important in the management of a variety of diseases. The age of the foal, primary disease process, and concurrent problems (i.e. hypotension, dehydration) impact the drug choice. The editors recommend the use of *N*-butylscopalammonium bromide (Buscopan[R]) as an initial choice in most cases. This anti-spasmodic frequently relieves signs of mild colic. NSAIDs are the first-line analgesics of many practitioners unless dehydration, hypotension or renal disease are present. Flunixin meglumine or ketoprofen are commonly used in foals of all ages.

If more potent analgesia is required, butorphanol alone may be effective in young foals. In older foals, concurrent administration of an $\alpha2$ agonist is recommended. Ideally, $\alpha2$ agonists should be used judiciously in the preoperative period to minimize adverse effects on blood pressure. Diazepam can be used for sedation of young foals; however it provides no analgesia.

Foals that are bloated may be administered neostigmine at a dose of 1–2 mg subcutaneously every 1–2 hours for 2–3 doses and then 4x daily thereafter. There may be a transient increase in colic signs following the administration of neostigmine.

Recommended reading

Freestone J 2002 Colic in foals. In: Mair T et al (eds) Manual of gastroenterology. Saunders, Philadelphia, p 2002

Lavoie JP, Drolet R, Parsons D et al 2000 Equine proliferative enteropathy: a cause of weight loss, colic, diarrhoea and hypoproteinemia in foals on three breeding farms in Canada. Equine Veterinary Journal 32:418–425

MacKay RJ 2001 Equine neonatal clostridiosis: treatment and prevention. Compendium on Continuing Education for the Practising Veterinarian 23:280–283

McAuliffe SB 2004 Abdominal ultrasonography of the foal. Clinical Techniques in Equine Practice 3(3): 308–316

Murray MJ 2002 Stomach diseases of the foal. In: Mair T, Divers T, Ducharme N (eds) Manual of equine gastroenterology. Saunders, Philadelphia, p 469–476

Paradis MR 2003 Nutritional support: enteral and parenteral. Clinical Techniques in Equine Practice 2(1): 87–95

Reef VB 1998 Pediatric abdominal ultrasonography. In Reef VB (ed) Equine diagnostic ultrasound. Saunders, Philadelphia, p 364–403

Slovis NM 2003 Gastrointestinal failure. Clinical Techniques in Equine Practice 2(1): 79–86

Sprayberry KA 2003 Gastric outflow obstruction in young horses. Current Therapy in Equine Medicine 5:101–104

Tillotson K et al 2002 Partial parenteral nutrition in equine neonatal clostridial enterocolitis. Compendium on Continuing Education for the Practising Veterinarian 24:964–969

Wilkins PA 2004 Disorders of foals. In: Reed SM et al (eds) Equine internal medicine. Saunders, St Louis, p 1381–1431

CHAPTER 5

The respiratory system

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Introduction

Respiratory disease in foals is one of the major causes of morbidity and mortality in the young horse. Early detection and treatment of respiratory problems are essential for not only the animal's wellbeing but also for the animal's athleticism in performance animals. Disorders of the respiratory system are second in importance only to those of the musculoskeletal system in limiting the athletic performance of the horse.

Diagnosis

Diagnostic approach to respiratory disorders should include:

- history taking including age, breed and environment
- physical exam:
  - auscultation of the chest
  - percussion of the chest
- endoscopy
- sinuscopy
- ultrasonography
- radiography.

Age and breed

Knowing the age and breed of the foal will provide insight as to the problem. Congenital problems are usually noted at birth while acquired diseases such as bacterial pneumonia tend to occur when the foal is older (>2 weeks of age).

The breed of the animal is important in determining the cause of the respiratory disorder. For example, Arabian horses with chronic lung infections should be evaluated for severe combined immunodeficiency syndrome (see Chapter 11, p. 293). Selective immunoglobulin M (IgM) deficiency tends to occur more frequently in Arabians and Quarter Horses, whereas agammaglobulinemia has been documented in Thoroughbreds and Standardbreds (see Chapter 11, p. 295).

Auscultation of chest (Figs 5.1 & 5.2)

Auscultation of the chest includes the use of either a re-breathing bag or covering the nares to cause hyperpnea. Bronchovesicular sounds in the young animal can be easily noted because of the decreased muscle mass and less attenuation of the lung sounds. Increased airflow will also increase the bronchovesicular sounds – especially in febrile, excited or hyperpneic animals.

However auscultatory findings do not always correlate with the degree of alveolar ventilation. Foals with lung consolidation may still have normal bronchovesicular sounds because of radiation of airflow from the adjacent areas. Therefore auscultation alone cannot be used to diagnose pathologic conditions of the lower airway.

Crackles may be auscultated and represent the equalization of pressure between two compartments (alveoli and the bronchioles) after the airways have opened (i.e. mucus in the airway that does not allow air to flow into the alveoli but when a deep breath is taken the airway expands allowing air flow past the mucus and into the alveoli).


Figures 5.1 & 5.2: Auscultation of the thorax with and without occlusion of the nares.

Wheezes are musical sounds that arise from vibrations within the airway walls by turbulent airflow noted during constriction of the airways.

Thoracic ultrasound (Figs 5.3–5.5)

Ultrasonographic examinations can be performed with a multifrequency 5.0- or 7.5-MHz linear transrectal transducer. The 7.5-MHz transducer will be able to display a depth of 4–12 cm, which is ideal for thoracic examination of a foal. Isopropyl alcohol is copiously applied to the hair coat to provide surface contact between the transducer and the foal. The alcohol helps to reduce the intervening trapped air. The thorax should be thoroughly scanned in a dorsal to ventral plane from the 16th to the 3rd intercostal space.

Sound waves are completely reflected at the normal aerated lung interface allowing only the pleural surface to be evaluated. Therefore, the normal visceral pleural edge of the lung appears as a straight hyperechoic line with characteristic equidistant reverberation air artifacts indicating normal aeration of the pulmonary periphery. The pleural edge of the lung is imaged gliding dorsally and ventrally when watching the patient breathe during thoracic ultrasonography. Only when fluid or cellular accumulation in the lung occurs immediately beneath the visceral pleural surface will an acoustic window be created, allowing visualization of pulmonary pathology. The affected area of the lung is hypoechoic and/or lacks the normal air echo at the surface.

It is critical to examine the lung carefully during both inhalation and exhalation. During inhalation air will enter the alveoli and surrounding airways resulting in reflection of sound waves. This reflection of the sound waves can prevent the visualization of pulmonary disease.

Pulmonary abscesses are variable in size and may be located anywhere in the lung. They are identified ultrasonographically by their cavitated appearance and the absence of any normal pulmonary

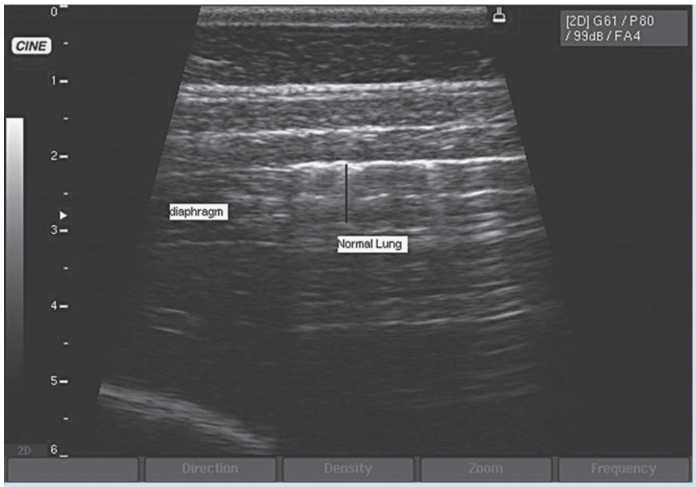


Figure 5.3: Normal ultrasonographic image of the thorax from a foal. Visceral pleural edge of the lung appears as a straight hyperechoic line with characteristic equidistant reverberation air artifacts indicating normal aeration of the pulmonary periphery (right = dorsal, left = ventral).

structures detected within the abscess. The center may appear hypoechoic, isoechoic or septated, depending upon the type of fluid present.

Pulmonary consolidation is hypoechoic and/or lacks the normal air echo at the surface.

Pleural effusion is characterized ultrasonographically as an anechoic space between the lung, thoracic wall, diaphragm and heart. Pleural effusions may be echogenic containing fibrin, blood, cellular debris and on occasion gas.

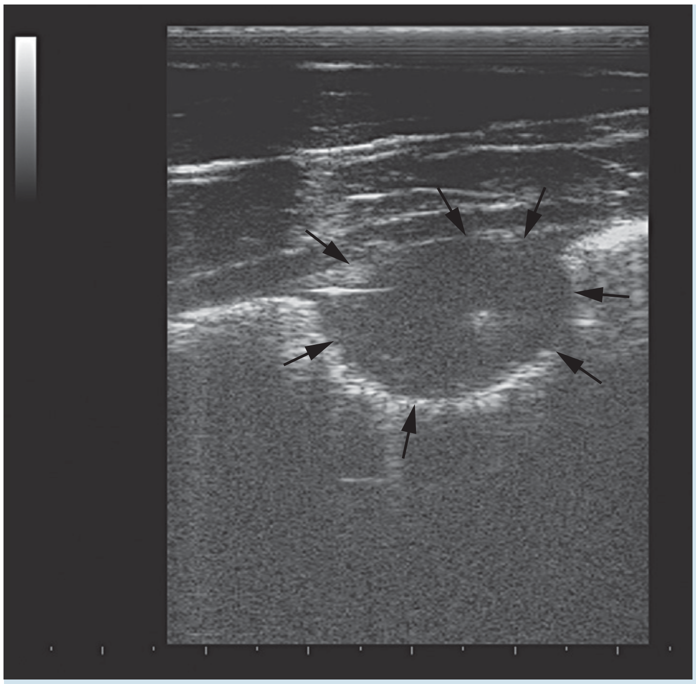


Figure 5.4: Abscess on the pleural surface in a foal diagnosed with *Rhodoccocus equi* pneumonia. The affected area of the lung is hypoechoic or lacks the normal air echo at the surface (arrows).

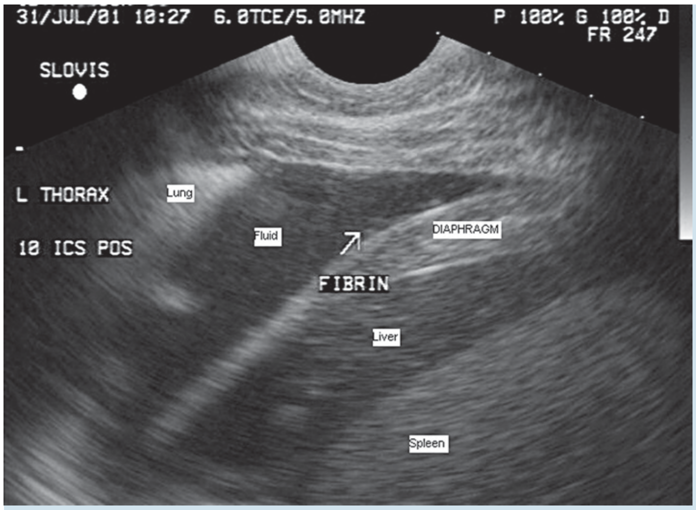


Figure 5.5: Pleural effusion. Note the hypoechoic fluid within the pleural cavity. Small hyperechoic "tags" are noted on the diaphragm. These "tags" are fibrin, which are characteristic of prolonged pleural pneumonia.

Radiography (Figs 5.6 & 5.7)

Radiography is considered by some to be the principal imaging technique for the evaluation of the thorax. However, radiographic examination has its limits (in the field and patient size) therefore ultrasonography is a valuable alternative in many cases.

Radiographs of the thorax in foals obtained within the first few hours of birth usually have a generalized interstitial opacity due to incomplete inflation. Within 12 hours the lungs become more lucent as the foal becomes active and the lungs completely inflate. The generalized opacities noted during the first few hours of life make radiography in neonates a diagnostic challenge. Any questionable abnormalities should therefore be re-evaluated in 24–48 hours. Additionally the cardiac silhouette will occupy a greater proportion of the thoracic cavity in the immature foal than in older animals.

Figure 5.6: Positioning for obtaining thoracic radiographs.

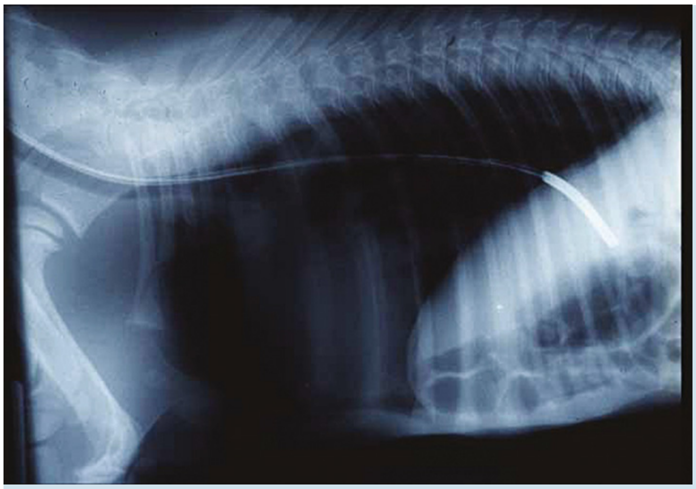


Figure 5.7: Normal newborn thoracic radiograph. The cardiac silhouette occupies a greater portion of the thoracic cavity in the neonate than in the adult. The thorax of a foal within the first few hours of birth may have a generalized interstitial opacity due to incomplete inflation. Within the first 24 hours the interstitial opacity resolves and the lungs become more lucent (more completely inflated). Foals with questionable abnormalities should be re-evaluated within 24–48 hours. The portions of the abdomen which are seen in this film are normal. Note the placement of the stomach tube.

Transtracheal wash (Figs 5.8–5.15)

A tracheal wash (TW) is a technique used to collect fluid from the lower respiratory tract for bacterial culture and cytologic evaluation. The results of the wash can help determine a proper treatment protocol for equine respiratory tract disease. Two techniques of obtaining a TW are illustrated: percutaneous and guarded endoscopic sampling.

Blood gas analysis

See Chapter 3 (p. 66) – "Assessment of hypoxemia and determining oxygen requirement" for further details.

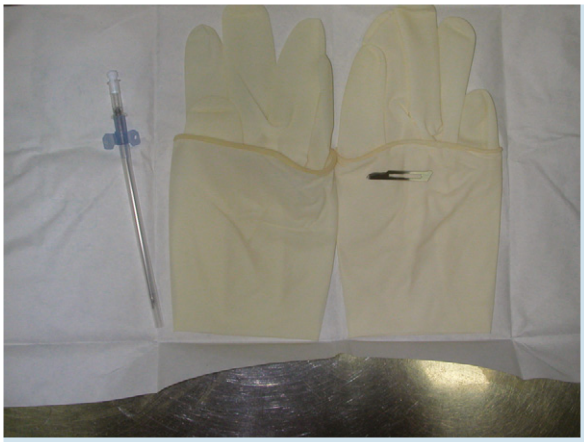


Figure 5.8: Tracheal wash kit. A variety of needle-catheter combinations can be purchased. Many veterinarians make their own kit which consists of a #11 or #15 scalpel blade, a 12-gauge over the needle cannula/catheter and a 6F (2.0 mm OD) × 50 cm dog urinary catheter with the tip cut off.

Figure 5.9: An area measuring 3 cm × 3 cm over the proximal third of the trachea is clipped and surgically prepared. Approximately 1 mL of lidocaine is infused subcutaneously.

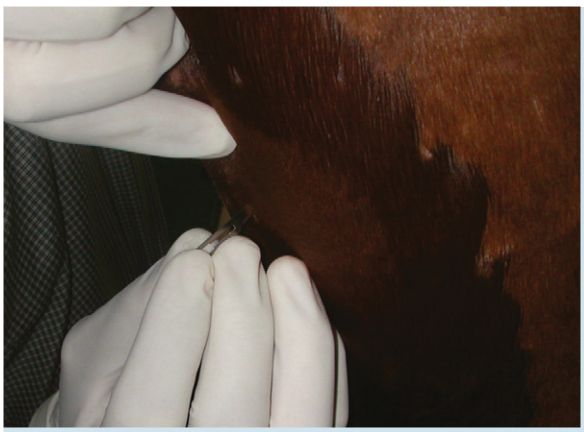


Figure 5.10: A small stab incision is made through the skin and subcutaneous tissue using a number 15 surgical scalpel blade.

Figure 5.11: The trachea is stabilized in one hand and the cannula is punctured through the trachea between two cartilage rings.

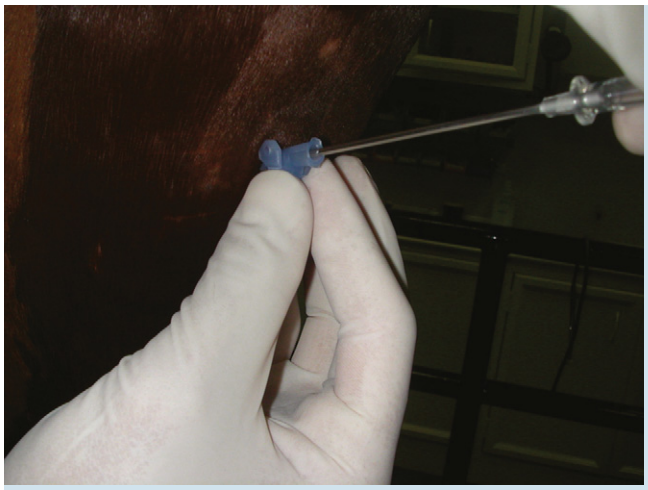


Figure 5.12: The stylet is removed and the urinary catheter is passed down into the tracheal lumen to the level of the thoracic inlet.

Figure 5.13: 30–35 mL of sterile saline is infused and then aspirated. No changes in relative cell counts occur in samples stored for 24 hours at 4°C in a capped syringe. If delays are expected to be longer than 24 hours then your local laboratory should be contacted to identify a proper fixative solution before the sample is sent for analysis.

Figure 5.14: Once the sample is obtained the catheter should be removed *before* the cannula. If the cannula is removed first then bacteria may be "dragged" into the peritracheal tissue causing an abscess – as noted in this foal.

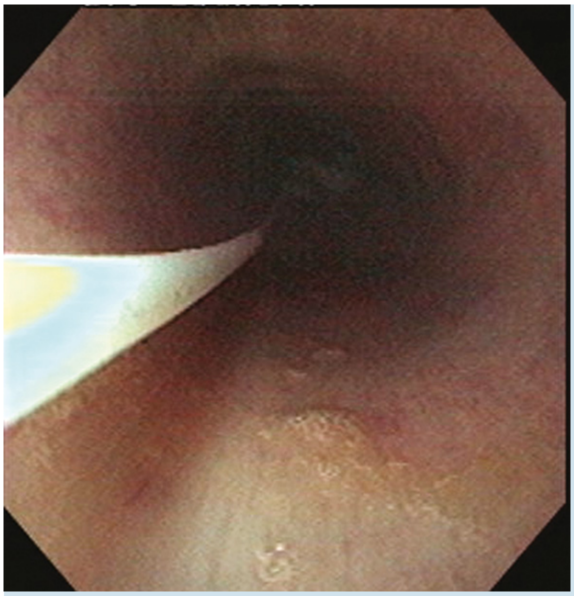


Figure 5.15: The guarded collection catheters are easy to use and reduce the incidence of complications associated with a percutaneous transtracheal wash. Contamination with this method is increased should the animal cough and have sputum touch the scope before aspiration. It is therefore necessary to have the patient properly sedated before the procedure.

Figure 5.16: Swelling over the right maxillary sinus in a 1-month-old foal with a maxillary sinus cyst. The foal had total occlusion of the right nasal passage. Note the epiphora from the right eye.

Figure 5.17: Lateral view of Figure 5.16. Percussion of the rostral and caudal compartments of the maxillary sinus revealed a dull resonance suggestive of a space occupying lesion.

Pleural fluid examination

See p. 164.

Congenital defects

Sinus cysts (Figs 5.16–5.18)

Sinus cysts are single or loculated fluid-filled cavities with an epithelial lining and can be found in horses of a wide age range. They develop in the maxillary sinuses and ventral concha and can extend into the frontal sinus.

Clinical signs

Clinical signs include: facial swelling, nasal discharge, dullness on percussion and partial/complete obstruction of the nasal passage.

Diagnosis

Radiographs are more helpful than endoscopic examination for diagnosis. A bony capsule may be distinctive.

Figure 5.18: Radiographs of the foal in Fig 5.16 were inconclusive for a sinus cyst because the entire maxillary sinus had a soft tissue opacity. A sinocentesis was performed of the rostral maxillary sinus to determine if the soft tissue opacity was secondary to fluid or a cyst. The rostral maxillary sinocentesis landmarks are 2.5 cm rostral and 2.5 cm ventral to the medial canthus of the eye. A 14 gauge 2.5 cm needle was used to penetrate the maxillary sinus. The sinocentesis revealed a mucohemorrhagic exudate in the rostral maxillary sinus. Repeat radiographs were taken after 100 mL of exudate were evacuated. Radiographs confirmed that a maxillary cyst was present and was occluding the nasomaxillary aperture causing excessive mucus to be present in the sinus. Surgical removal of the cyst and involved conchal lining was performed. No further problems were noted after the surgery.

Treatment

Surgical removal of the cyst and involved conchal lining.

Choanal atresia (Fig 5.19)

This is a rare disorder that is due to a failure of the breakdown of the buconasal membrane. This membrane normally separates the embryological buccal and nasal cavities and breaks down during later stages of development. There is therefore no communication between the oropharynx and the nasopharynx.

Clinical signs

- Bilateral atresia results in immediate respiratory distress, cyanosis and decreased/absent airflow from the nares following birth.
- Unilateral atresia results in a lack of airflow through the affected nasal passage.
- Passage of a nasogastric tube will be obstructed at the pharynx.

Diagnosis

Endoscopic examination will reveal the obstruction in the posterior nares.

Treatment

Immediate tracheostomy is warranted during respiratory distress. Surgical intervention has been described with the prognosis for athletic performance being described as poor.

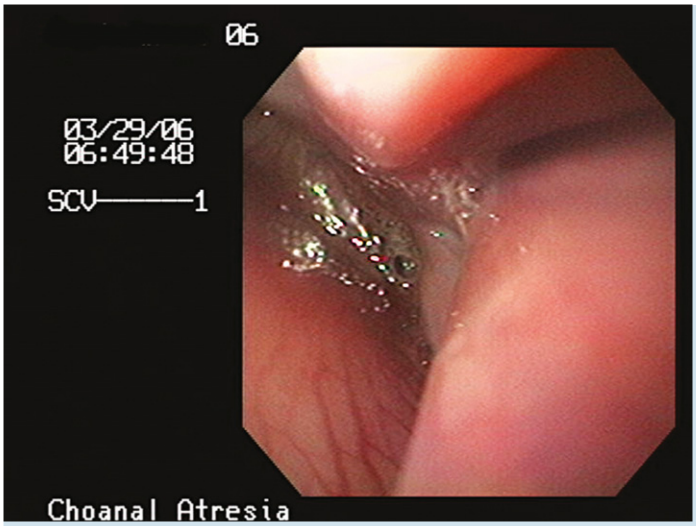


Figure 5.19: Choanal atresia in a newborn foal that presented in respiratory distress. Orotracheal intubation was performed at the farm to aid in ventilation. Endoscopy revealed complete obstruction of the left and right posterior nares.

Guttural pouch tympany (Figs 5.20–5.28)

Guttural pouch tympany is usually unilateral but can be bilateral and is more common in fillies than in colts. The etiology is not exactly known but it appears that the salpingopharyngeal fold (redundant tissue) is excessive, causing the external ostia to act as a one-way valve. This one-way valve action will cause the accumulation of air in the guttural pouch.

Clinical signs

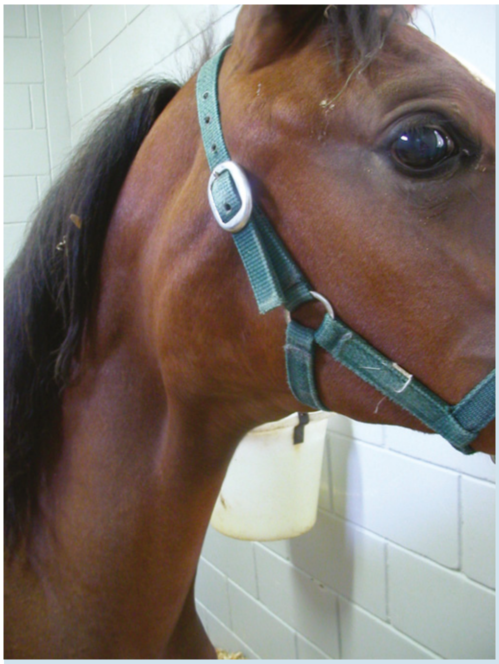
- The affected pouch is distended with air to form a non-painful, elastic swelling in the parotid region.
- The swelling is most prominent on the affected side, but can extend across the neck and give the impression of bilateral involvement.
- Development of this lesion can occur soon after birth or weeks later.
- Distension of the affected pouch(es) causes pharyngeal compression resulting in sterterous airflow.

Diagnosis

- Clinical appearance is typical.
- Skull radiographs are diagnostic, but endoscopy will be warranted to confirm involvement of one or both ostia.
- *If in doubt as to the involvement of one or both pouches, air can be released from one pouch by either placing a blunt probe through the ostia on one side or aspirating air percutaneously on one side. If this results in complete resolution of the swelling then only one pouch is affected.*

Treatment

- The placement of an indwelling Foley catheter into the affected guttural pouch(es) can offer temporary relief. This can be done via Viborgs triangle or via the ipsilateral salpingopharyngeal ostium.
- For severe cases of pharyngeal collapse the use of a tracheostomy prior to surgery will be warranted.



Figures 5.20 & 5.21: Arabian weanling with unilateral guttural pouch tympany seen before and 3 months after fenestration surgery.

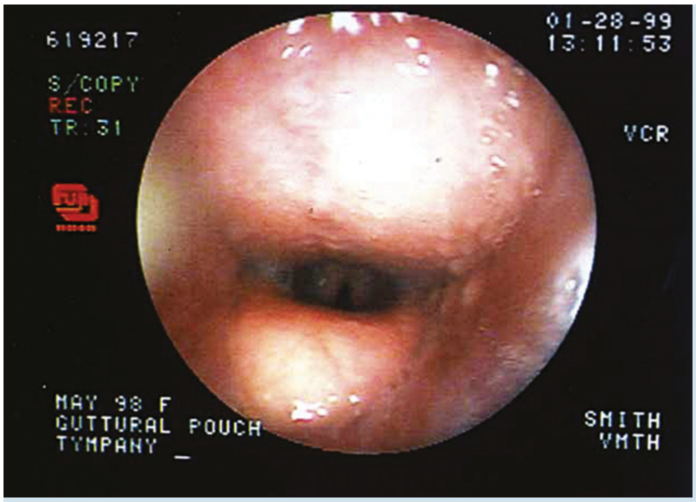


Figure 5.22: Pharngoscopy of the foal in Figs 5.20 and 5.21. Note the marked pharyngeal collapse secondary to the guttural pouch tympany. Pharyngeal collapse appears similar with unilateral and bilateral tympany.

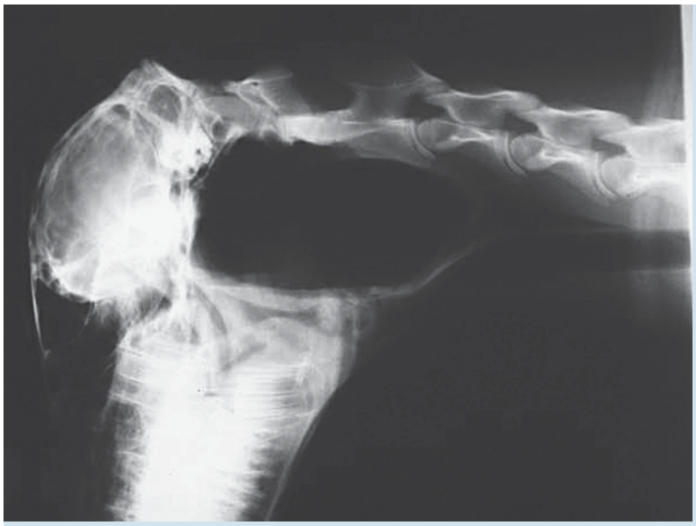


Figure 5.23: Radiograph of a foal with bilateral guttural pouch tympany. Note the characteristic marked air distension of the guttural pouches. Compare with normal guttural pouches in Fig 5.30.

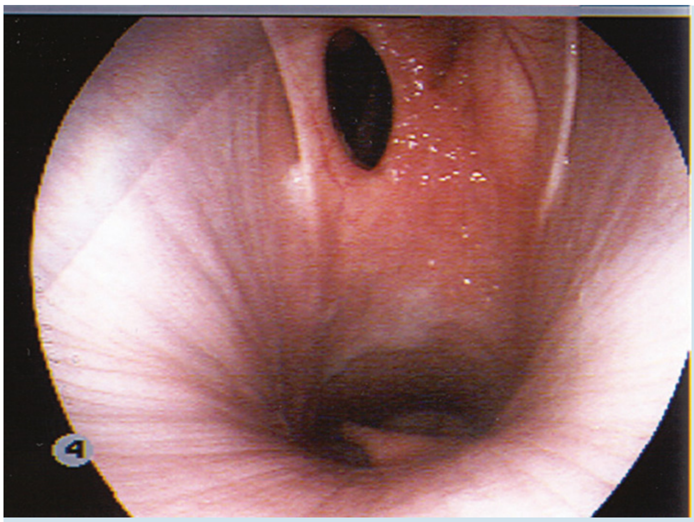


Figure 5.24: Fistulation into the guttural pouch through the pharyngeal recess avoiding the potential of nerve damage. This foal did well after surgery.

Figures 5.25 & 5.26: Arabian foal with bilateral guttural pouch tympany. Note the massive swelling in the parotid areas. This filly required an emergency tracheostomy.

- For unilateral tympany a fenestration through the median septum is achieved either via endoscopic-guided laser or blunt dissection via a hypovertebrotomy incision which will allow the egress of trapped air from the tympanitic pouch through the normal side.
- With bilateral involvement transendoscopic laser fenestration of the septum or fistulation into the guttural pouch through the pharyngeal recess avoids a surgical incision and the risk of nerve damage.

Branchial cyst (Figs 5.29 & 5.30)

Branchial cysts are uncommon embryonic anomalies of the horse. The cysts result from malformation of one of the five branchial arches during embryogenesis.

Figure 5.27: Foley catheter placed in the guttural pouch via Viborg's triangle for temporary relief.

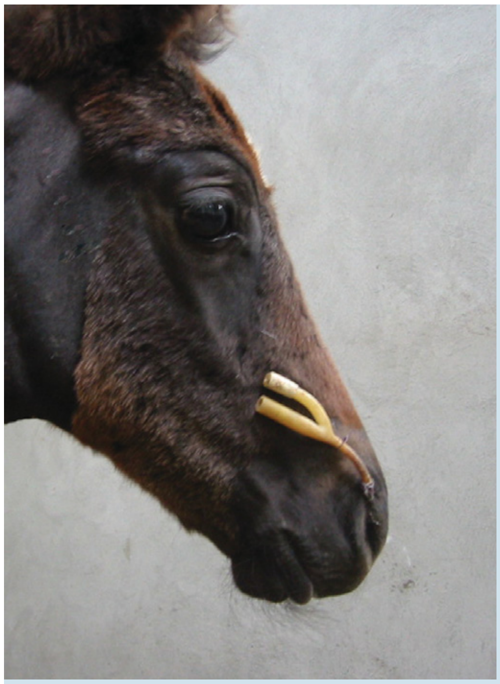


Figure 5.28: Foley catheter placed in the guttural pouch via the ipsilateral salpingopharyngeal ostium and fixed in place by sutures at the nares.

Clinical signs

- Typically appear as smooth round mobile masses and often do not cause any clinical abnormalities, although large cysts may cause discomfort and can disrupt pharyngeal or laryngeal function.
- Cysts may not be apparent until weaning age. The late recognition of branchial cysts may be a result of delayed secretion of fluid by the epithelial lining.

Figure 5.29: Branchial cyst in a 4-month-old Morgan weanling. The weanling had a smooth round mobile mass located in the right parotid area.

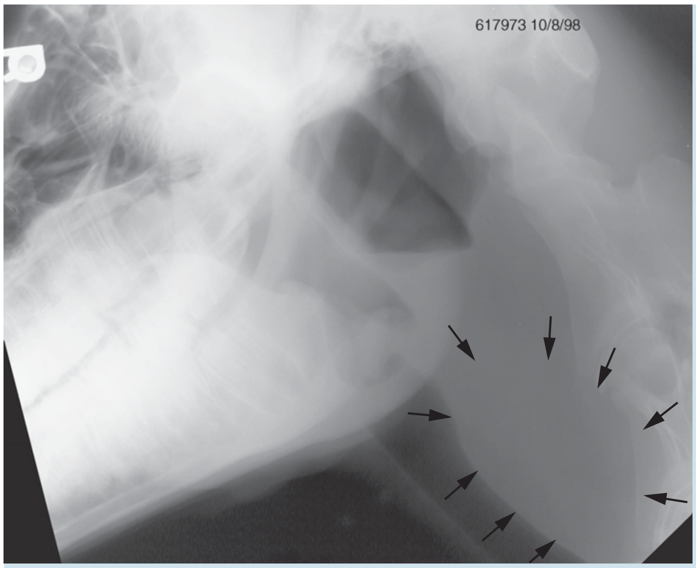


Figure 5.30: Lateral radiographic view of the skull and cranial cervical region of a weanling with a branchial cyst. Note the large space occupying soft tissue mass ventral to the 2nd cervical vertebra (arrows) that is causing ventral displacement of the trachea. Note also the normal size of the guttural pouches as compared to Fig 5.23.

Diagnosis

- The clinical appearance of the branchial cysts may be very similar to guttural pouch tympany and enlarged retropharyngeal lymph nodes.

Figure 5.31: Subepiglottic cyst in a 6-month-old Saddlebred with a 3-month history of coughing and stertorous breathing.

- Endoscopic examination may reveal collapse of the dorsal pharyngeal wall and compression of the ipsilateral lateral compartment of the guttural pouch.
- Ultrasound would reveal an anechoic fluid-filled structure with a well defined hyperechoic capsule. Aspiration of the cysts would reveal an amber viscous fluid with a high protein content and a low cellularity (lymphocytes and macrophages).
- Radiographs would reveal a large space-occupying soft tissue mass.

Treatment

- Medial management would consist of marsupialization and iodine sclerotherapy.
- Surgical excision may be necessary for larger cysts.

Epiglottic and pharyngeal cysts (Figs 5.31–5.33)

Epiglottic and pharyngeal cysts are fluid-filled structures that do not communicate with an epithelial surface. These cysts are usually found in the subepiglottic tissues, in the dorsal nasopharynx and within the soft palate. In foals these cysts are considered congenital and depending on their location may represent embryonic remnants of the thyroglossal and craniopharyngeal ducts. Thoroughbred and Standardbred foals are most commonly affected.

Clinical signs

- Foals with large cysts may present in respiratory distress which requires a temporary tracheostomy.
- Coughing, nasal discharge, nasal milk reflux, and aspiration pneumonia are common clinical features.

Figure 5.32: Diode laser ablation of the cyst in Fig 5.31.

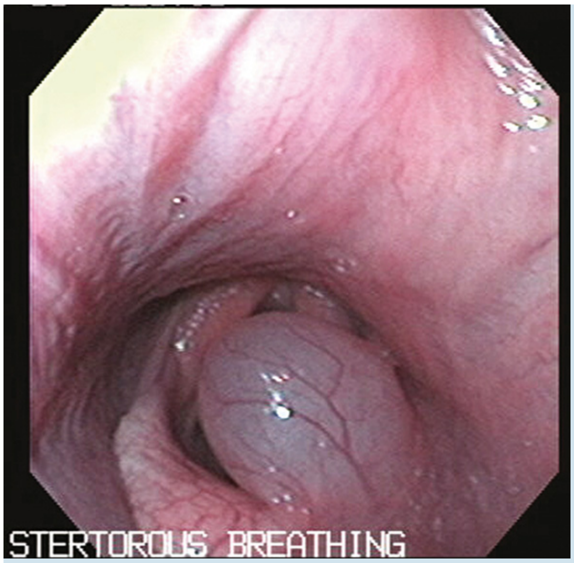


Figure 5.33: Aryepiglottic congenital cyst. 3-hour-old foal with a history of stertorous respiratory pattern.

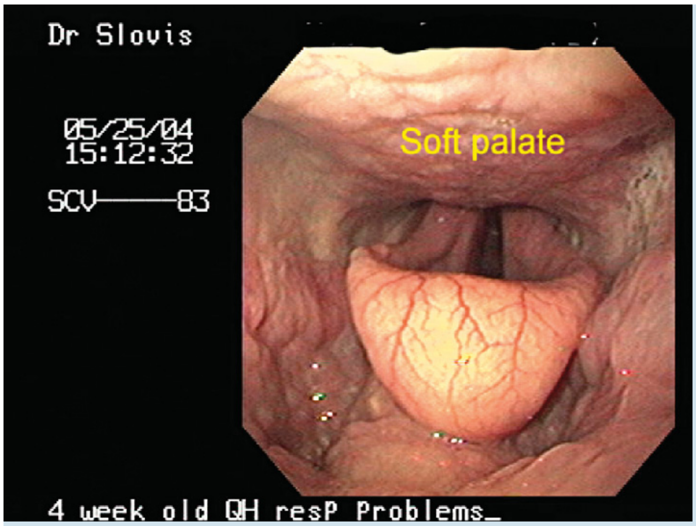


Figure 5.34: Oropharyngeal view of a dorsal displacement of the soft palate from a 4-week-old QH with sterterous breathing. Note the ventral curvature of the epiglottis.

Diagnosis

Endoscopy is essential for diagnosis. Make sure that swallowing is observed during endoscopy to allow observation of the ventral aspect of the epiglottis and the caudal aspect of the soft palate.

Treatment

Surgical intervention will be warranted. This would include either the use of laser ablation or surgical resection via a ventral midline laryngotomy.

Restriction of the glossoepiglottic fold (Figs 5.34 & 5.35)

The glossoepiglottic fold is a normal structure on the ventral aspect of the epiglottis consisting of mucosa, adventitial tissue and skeletal muscle. It is unknown if the muscle is dysfunctional or just restrictive. This fold at one time was thought to be a persistent epiglottic frenulum described in literature. There have been a handful of cases that have a persistent dorsal displacement of the soft palate which have responded favorably to transection of the glossoepiglottic fold. More research is needed to help characterize this disorder.

Clinical signs

- Foals are usually admitted with a complaint of oronasal reflux after nursing.
- Variable systemic signs of aspiration pneumonia may be evident.

Treatment and differentials

- If identified a very young foal (<1 week of age), conservative therapy with NSAIDs and vitamin E is recommended. Enteral feeding via

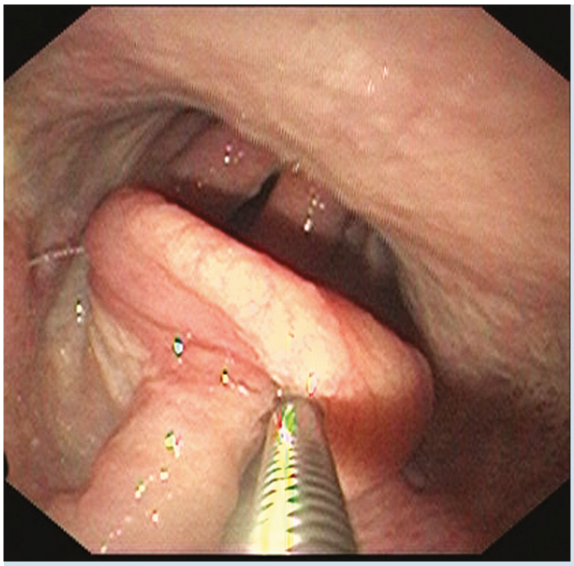


Figure 5.35: A blunt probe is used to lift the epiglottis and reveal the glossoepiglottic fold. This fold was pale and thin compared to normal. The glossoepiglottic fold was resected resulting in normal anatomical positioning.

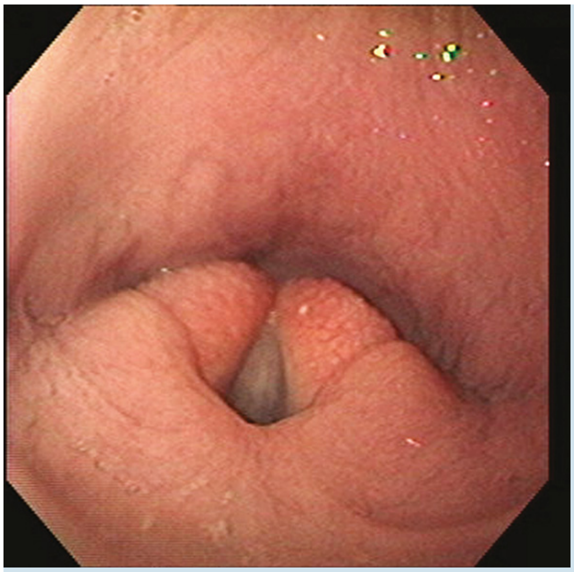


Figure 5.36: Soft palate elongation. Note the dorsal displacement of the soft palate (DDSP) and the caudal elongation of the soft palate. The foal had marked respiratory stertor. A tracheostomy was performed to help reduce the turbulent airflow in the URT. The soft tissue edema of the soft palate not resolve with the tracheostomy or medical therapy. A staphylectomy was performed to excise the redundant tissue and correct the DDSP.

a nasogastric tube will also be required to avoid aspiration, and antibiotics should be given if there is any evidence of an existing aspiration pneumonia. The author has seen foals with pharyngeal dysfunction as a result of perinatal asphyxia that self corrected over 2–3 weeks.

- If edema is noted of the soft palate causing occlusion of the rima glottis then a temporary tracheostomy will be needed to help decrease the turbulent airflow and excessive negative pressure in the upper respiratory tract (URT), especially when the animal develops tachypnea and respiratory stertor. The tracheostomy may have to be in place for 10–14 days until the nuerogenic disorder arising from perinatal asphyxia has resolved.
- Surgical resection of the glossoepiglottic fold is indicated if the animal has not responded to conservative therapy.
- Surgical resection of a restrictive glossoepiglottic fold involves either sharp transection or transection with a laser. Once corrected these foals tend to recover very well.

Soft palate elongation (Figs 5.36–5.38)

This disorder is rarely noted. Foals born with this disorder tend to have a persistent dorsal displacement of the soft palate (DDSP) and what appears to be an excessive length of the soft palate. The affected foals only show signs of distress when stressed or running around.

Clinical signs

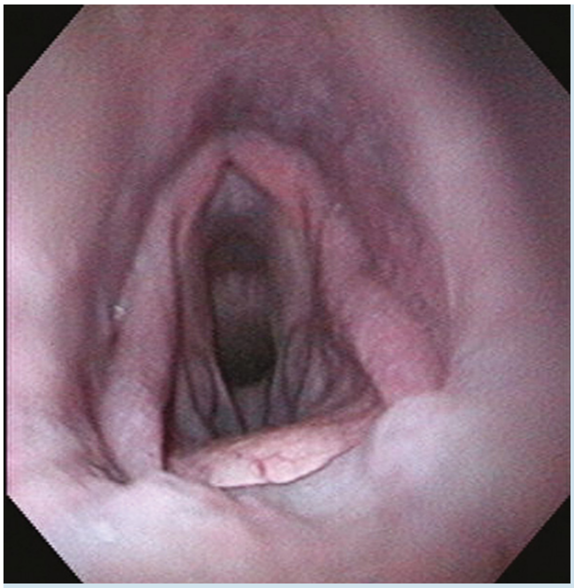
- Sterterous breathing with excessive coughing and/or milk discharge from the nares. These findings are not consistent but usually are associated with exercise or stress when nursing.
- Aspiration pneumonia is a common finding.

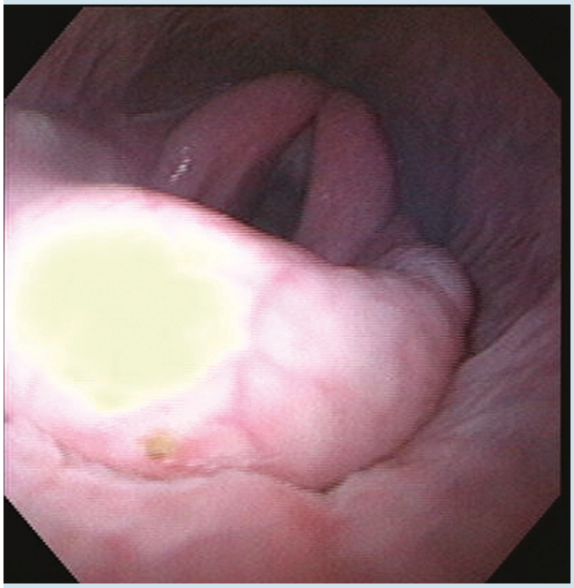
Diagnosis and differentials

- Pharyngoscopy will reveal a DDSP and a soft palate that appears elongated and edematous.
- It is important to rule out a neurological disorder of the pharynx before treatment (e.g. perinatal asphyxia).
- It may be hard to distinguish this condition from a restriction of the glossoepiglottic tissue.
    - Examination of the oropharynx and the use of a blunt probe to help lift the epiglottis into a "normal" anatomical position may help determine if the animal has restriction of the glossoepiglottic tissue. If the epiglottis cannot move dorsally toward the soft palate then a restriction of the glossoepiglottic fold would be suspected.

Treatment

- Conservative treatment should be initiated first which consists of NSAIDs, vitamin E (5000 IU PO SID), fluids with DMSO and when warranted the placement of a nasogastric feeding tube.
    - Conservative treatment may take 2–3 weeks before there are any noticeable changes
- Staphylectomy is indicated ***only*** if the animal does not respond to conservative treatment.





Figures 5.37 & 5.38: 6 week re-check post staphylectomy. The foal has been running in the pasture and nursing with no evidence of respiratory compromise. Note that the epiglottis could be visualized and there is resolution of the DDSP. 2-year follow up indicated a normal appearance of the epiglottis and soft palate prior to training.

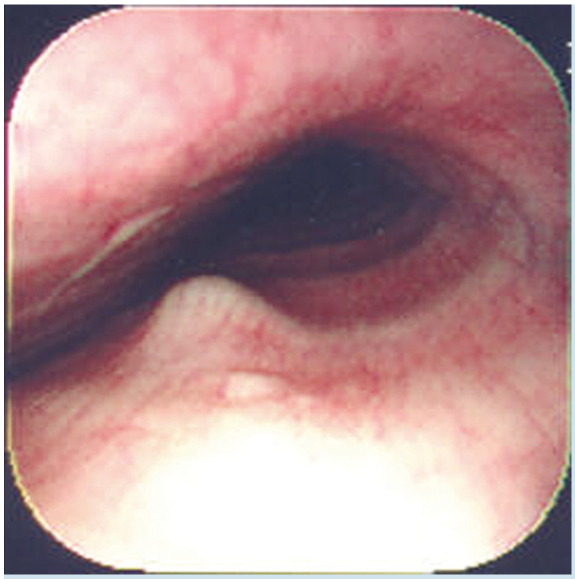


Figure 5.39: Severe collapse of the trachea and ring malformation in a miniature foal. Note the increased dorsoventral flattening of tracheal lumen.

Tracheal collapse (Fig 5.39)

Tracheal collapse might be related to abnormal tracheal cartilage matrix and is most often diagnosed in ponies and miniature horses.

Clinical signs

- Typically signs are not noted until the animal is older and is often not noted until adulthood.
- Severely affected animals will develop a "honking" inspiratory and expiratory sound.
- Tracheal stenosis and collapse can also develop dramatic clinical signs, including respiratory stridor, dyspnea and cyanosis.

Diagnosis

The diagnosis of tracheal stenosis and collapse is based on signalment, history, clinical signs, endoscopic and radiographic findings.

Treatment

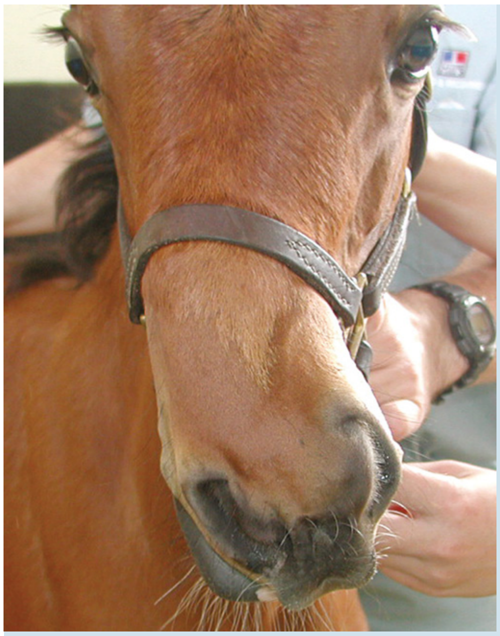
- Treatment of tracheal collapse has been reported with the use of an intraluminal stent in a 7-month-old miniature horse.
- No other significant treatment plans have been implemented in older foals to increase the success of surgical options.
- The prognosis is usually good for life but poor for athletic function.

Wry nose (Figs 5.40–5.43)

Wry nose, or deviated rostral maxilla and associated nasal septal deviation, is a congenital deformity in the horse. The deviation can be very mild to severe. Foals generally have a shortened maxilla along with the deviation. A foal with wry nose will have the upper jaw and nose deviated or turned to one side. A deviated nasal septum (the cartilage plate that separates the right and left nasal passageways) is also usually present, which results in obstruction of the airway and difficulty breathing. This is the greatest *functional* concern with wry nose. There will usually be malocclusion (poor alignment) of the teeth, although most foals can still nurse and in most cases are bright and active.

The etiology of maxilla deviations is often unknown. Failure of proper embryologic development of the hard palate and maxilla may be associated with possible genetic defects. Abnormal in-utero positioning has also been proposed.

Wry-nosed foals might result in dystocia, and can also have other deformities of the neck and occasionally of the limbs. It is very important to further evaluate the foal for other types of genetic defects. Examination of the hard and soft palate is very important to ensure a cleft is not present.



Figures 5.40 & 5.41: Wry nose.

Figure 5.42: Radiograph of a wry nose.

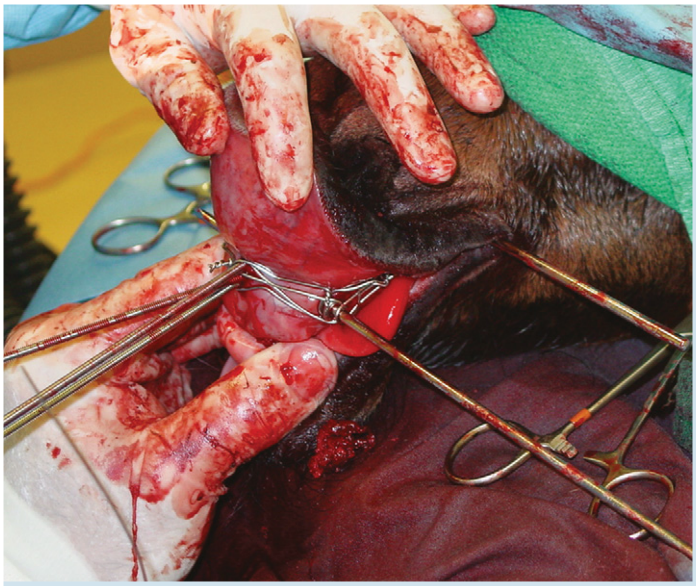


Figure 5.43: Intraoperative image of wry nose surgery. Note the pins and wires placed in the maxilla which was fractured and re-positioned.

Diagnosis

- Visual examination – obvious facial deviations
- Digital examination
- Oral examination
- Radiology – helps confirm and further evaluate the degree and severity of the deviation
- Endoscopic examination – helps assess the deviation and evaluates the hard and soft palate.

Treatment

Conservative management

Mild cases can potentially survive and live without significant airway problems.

Surgical management

In more severe cases surgical correction is generally undertaken in multiple stages. This type of reconstructive surgery is expensive and requires significant aftercare. Although the objective of the surgery is usually to make the horse capable of being an athlete, unfortunately, neither the functional or cosmetic outcome can be guaranteed.

- Surgical correction of moderate to severe cases has been described. The most commonly reported technique involves transecting both the left and right sides of the maxilla. The maxilla is positioned in

as correct an alignment as possible. The maxilla can be stabilized using pins or small reconstructive plates. The gap created on the concave side of the maxilla can be filled with a harvested section of rib or allograft. To further correct the deviation, the cartilaginous nasal septum that is cranial to the deviation is surgically removed. This can be removed at the same time as aligning the maxilla or at a later date. After correction, foals still are not cosmetically normal. The maxilla is often shortened.

- External fixation of the deviated maxilla has been described. This involved the same principles as an olizerof fixation device to elongate bone.
- Surgically creating a cutaneous fistula into the nasal passageway on the convex side can be performed.
- Permanent tracheostomy can also be performed.

Acquired diseases

Respiratory distress syndrome (Figs 5.44–5.48)

Respiratory distress syndrome is poorly understood and results in a failure of normal gas exchange at the alveolar level. This failure of proper gas exchange results in atelectasis (lack of pulmonary expansion).

Pulmonary immaturity and surfactant deficiency are the main causes of neonatal respiratory distress syndrome in humans. Surfactant dysfunction is primarily associated with immaturity of the lungs and is therefore commonly associated with premature foals. Full-term foals could also be affected – especially those with systemic disease, meconium aspiration, as well as viral and bacterial pneumonia which can result in altered or inappropriate surfactant production.

Persistent fetal circulation and cardiac congenital anomalies are examples of non-respiratory causes of respiratory distress syndromes seen in the newborn foal.

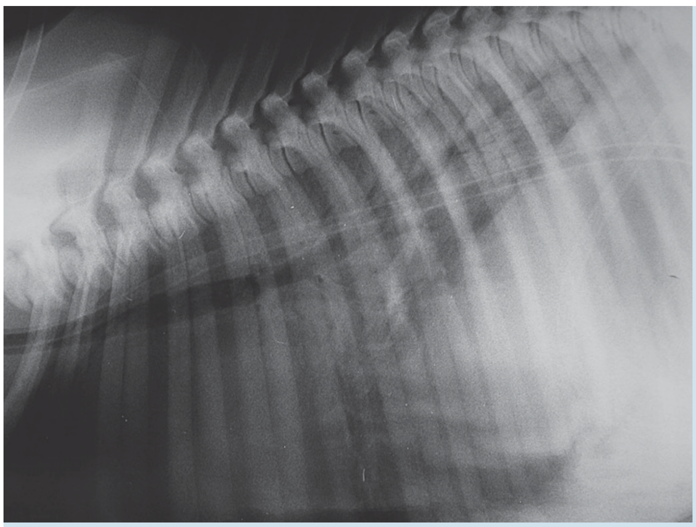


Figure 5.44: Prominent airbronchogram in a foal suffering from respiratory distress syndrome.

Clinical signs

- Animals present with flared nostrils, tachypnea, dyspnea and marked abdominal lift with inspiration.
- Mucus membranes are injected and if severe disease is present may be cyanotic.
- Before an animal goes into respiratory failure a paradoxical respiratory pattern will be noted. Paradoxical respiratory pattern is characterized by a downward motion of the diaphragm and consequent outward movement of the flanks without excursion of the rib cage during inspiration. This pattern indicates complete fatigue of the respiratory muscles.

Diagnosis

- Arterial blood gas analysis is an essential diagnostic tool when evaluating animals with respiratory distress syndrome. Hypoxemia

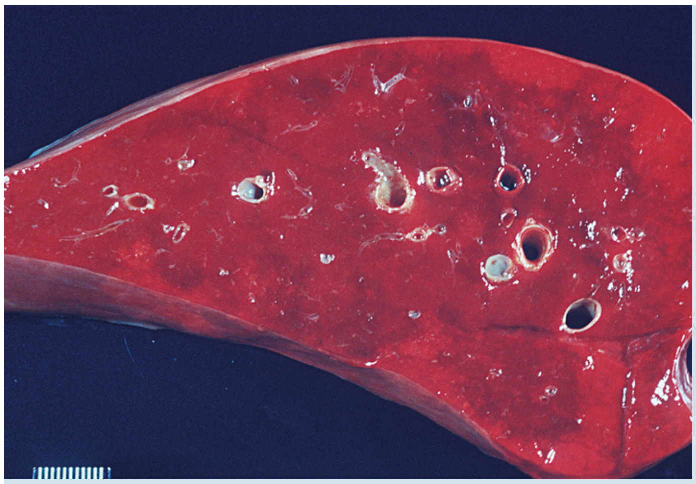


Figure 5.45: Markedly consolidated lung lobe. This section of lung did not float in water. This foal presented for surfactant deficiency and was in respiratory distress upon arrival. Same patient as in Fig 5.44.

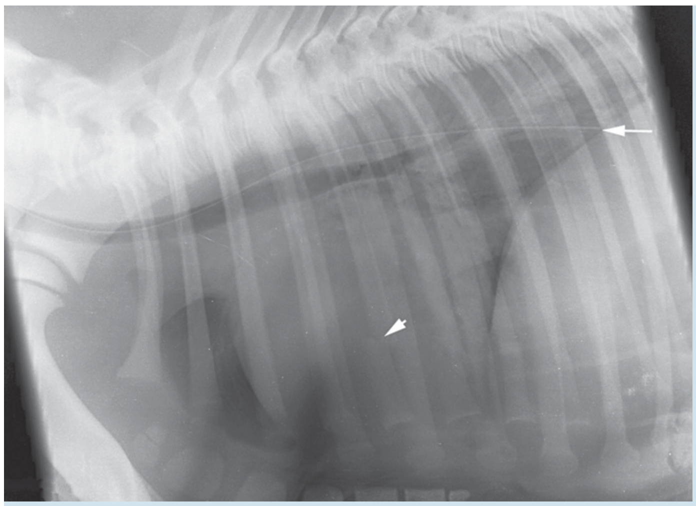


Figure 5.46: A lateral thoracic radiograph from a 30-hour-old Thoroughbred foal with respiratory distress syndrome. There are numerous airbronchograms reflecting atelectasis. The foal also has a fracture of the right fifth rib (arrowhead). Note an indwelling radiopaque nasogastric feeding-tube is visible (arrows).

Figure 5.47: Placement of an intranasal 22 French nasal catheter. A Harris Flush enema tube was used as a nasal catheter. Note that the catheter was sutured to the nares.

Figure 5.48: Severe cyanosis (PaO_2 of 55 mmHg) in a foal in respiratory distress syndrome secondary to prematurity and decreased surfactant production.

(i.e. partial pressure of oxygen (PaO_2) <80 mmHg) is a characteristic finding with this disease. $PaCO_2$ will be variable because carbon dioxide diffuses more readily than oxygen.

- Thoracic radiography is also important in the evaluation of these foals and reveals a hazy, granular appearance associated with interstitial or alveolar patterns. Prominent airbronchograms are usually noted.

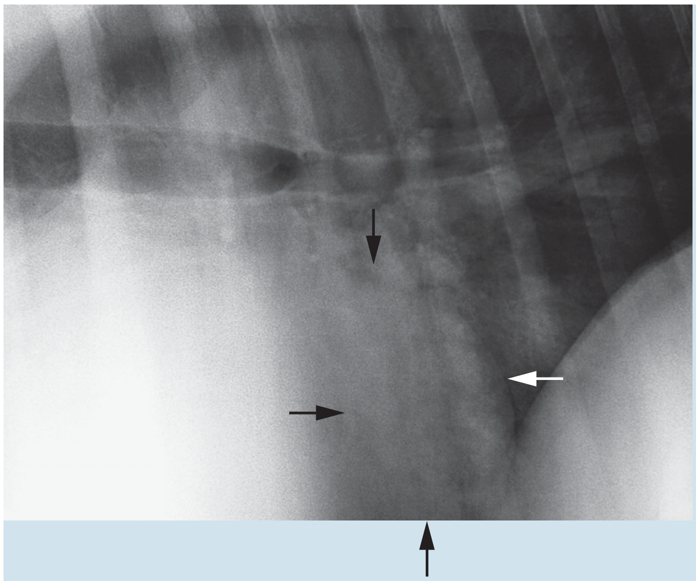


Figure 5.49: Radiograph of a foal that had meconium aspiration. Note the caudoventral pneumonia (increased radiodensity of the lung field just caudal to the cardiac silhouette) (arrows).

Treatment

- Therapy is aimed at preventing atelectasis, correcting hypoxemia and preventing hypercapnia.
- Re-positioning the foal into sternal recumbency allows better chest-wall expansion and respiration. This alone may increase PaO_2 by 10–20 mmHg.
- Nasal oxygenation at a rate of 6–15 L/min can help increase the fraction of inspired oxygen. If oxygenation insufflation and emergency bronchodilator therapy do not elevate the PaO_2 then assisted ventilation will be necessary.
- Vasorelaxants and bronchodilators may also be administered in conjunction with oxygen therapy to improve pulmonary function:
  - sildenafil 0.5–2.5 mg/kg PO SID
  - aminophylline 5–10 mg/kg IV QID (diluted in fluids)
  - corticosteroids – Solu-Delta-Cortef 1 mg/kg IV SID/BID or dexamethasone 0.1 mg/kg IV SID to BID.
- Surfactant replacement therapy. Should be given within 30 minutes of delivery for optimal success. The total dose is divided into four equal amounts and administered via the endotracheal tube with a long catheter. Each fourth should be administered with the foal placed in different positions (i.e. left lateral, dorsal, right lateral and ventral recumbency). The foal should be manually ventilated during and after surfactant administration. Typical dosage is 100 mg/kg.

Meconium aspiration (Figs 5.49 & 5.50)

In utero passage of meconium could be normal or occur during hypoxia (see Fig 2.66). In a hypoxic–ischemic event fetal reflex redistribution of cardiac output away from less vital organs such as the bowel results in intestinal ischemia followed by transient hyperperistalsis, anal sphincter relaxation, and meconium passage.

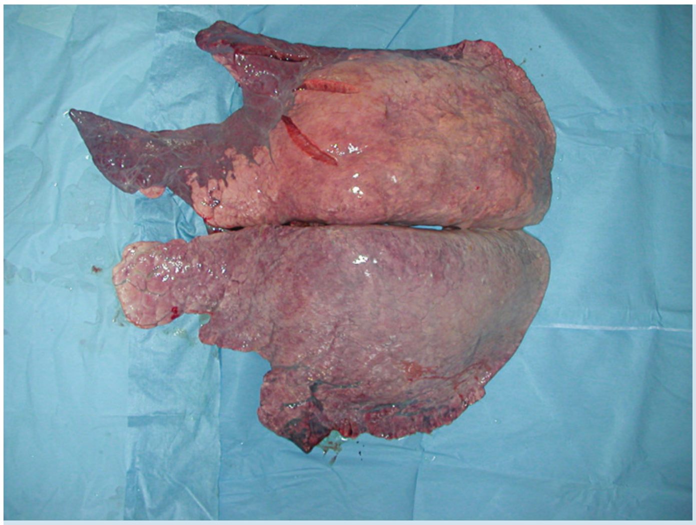


Figure 5.50: Gross pathology revealing marked consolidation (reddish purple discoloration) of the cranioventral right lung lobe. These lesions interfere with gas exchange resulting in ventilation/perfusion mismatch leading to hypoxemia. The foal died from severe sepsis secondary to the meconium aspiration.

During periods of stress the fetus may start gasping for breath and inhale the contaminated amniotic fluid, resulting in severe pneumonia. Meconium is very irritating to the lower airway initiating bronchoconstriction and pulmonary edema, which results in ventilation–perfusion mismatch.

Clinical signs

- Foals will be meconium stained with red/brown ocular and nasal secretions.
- Crackles and wheezes or tubular lung sounds may be auscultated in all airways.
- Marked abdominal lift may be noted with every expiration and inspiration of air.

Diagnosis

- Obvious clinical signs.
- Thoracic radiography will reveal cranioventral consolidation.
- Blood gas analysis will be characterized by a profound hypoxemia.

Treatment

- Initially because of the severe inflammation that may occur with meconium aspiration antibiotic therapy would be imperative.
- Corticosteroids may also be warranted to help prevent an acute respiratory distress crisis. Nasal oxygen supplementation (6–8 L/min) is usually initiated.
- Mechanical ventilation is usually unrewarding.

Equine herpes virus Type 1 and Type 4 (Fig 5.51)

See Chapter 1 p. 11, Chapter 10 p. 279. EHV-1 and EHV-4 are α-herpes viruses that are enzootic in most horse populations. Most foals experience EHV-1 and/or EHV-4 infections early in life. Once the animal is infected via direct horse-to-horse contact or inhalation of aerosolized virus the virus may establish latency. Latency sites for EHV-1 have been identified in neuronal cells of the trigeminal ganglia, peripheral blood lymphocytes, and lymphoid tissues. EHV-4 latency has been noted in lymphoid tissue, trigeminal ganglia and pulmonary epithelial cells. Newborn foals with EHV are most commonly exposed at or before birth which usually causes the mare to abort or give birth to a weak foal.

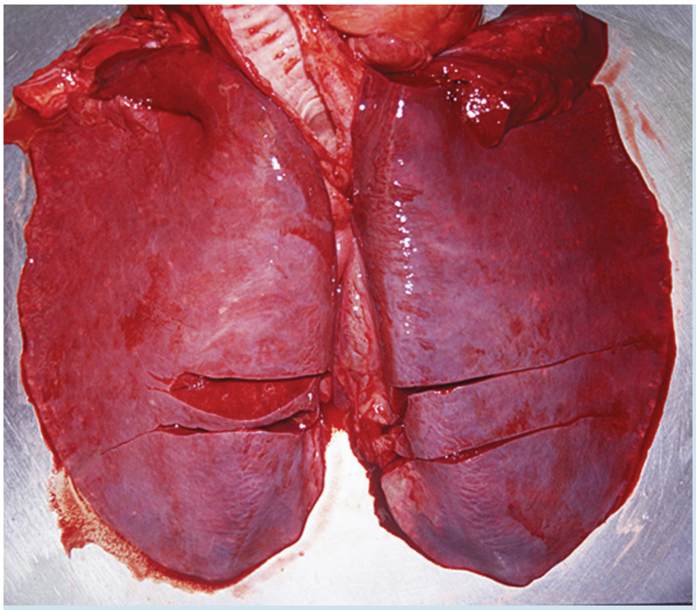


Figure 5.51: Severe atelectasis of a foal diagnosed with EHV-1. Note the small white necrotic foci of the lung parenchyma. Histopathology confirmed equine herpes virus lung inclusion bodies.

Clinical signs

- Newborn foals may be stillborn or very weak with death frequently occurring in 3 days. Should the foal live past 3 days it is usually weak with pneumonia, depressed or convulsing.
- Older foals will have initial signs including fevers, depression, lethargy, anorexia, lymphadenopathy, serous nasal discharge and coughing. The incubation period is 3–10 days with clinical signs persisting for 2–7 days.

Diagnosis

- *Neonates*. Ante mortem diagnosis is difficult. Buffy coat could be assessed for EHV-1/4 PCR and virus isolation. Liver and lung biopsy could help identify the infectious agent. Profound leukopenia is characteristic of foals with EHV-1 infection.
- *Older foals*. Preliminary diagnosis can be made based on the rapid spread of the disease and the presence of compatible signs. Hematological findings are inconclusive. Virus isolation and PCR assessment of pharyngeal swabs and buffy coat. Acute and convalescent titers (3 weeks after the acute febrile stage) can be assessed.

Treatment

This disease in foals is usually hopeless although on occasion a foal may survive. However, it may be difficult to differentiate from other

neonatal conditions, such as severe sepsis, and as a result many foals with neonatal forms of EHV-1 receive aggressive therapeutic support including fluids, total parenteral nutrition, nasogastric feedings and antiviral medication (valcyclovir 30 mg/kg orally q12h for 7–10 days may be of some benefit). Severely leukopenic foals may be administered granulocyte stimulating factor, Neupogen[R] 300 µg SQ (frequency depends on the response but normally only a single dose is given). The response is generally poor in severely affected foals.

Prevention

Vaccination appears to be the best method of controlling this infection. Current vaccinations do not prevent the infection with EHV-1/4, but they can help reduce the severity of signs, viremia and attenuation of viral shedding to help protect the herd. Foals usually start their vaccine program at 4–6 months of age with two doses 3–4 weeks apart and then the third booster 8–12 weeks later.

Mares are vaccinated with a commercially approved vaccine at 5, 7 and 9 months of gestation in order to prevent fetal infection.

Institution of an isolation program for incoming horses as well as a stall disinfecting program can help reduce the spread of this endemic disease.

Influenza

Equine influenza virus (EIV) is a major cause of equine respiratory disease. Equine influenza is a species type A influenza virus from the orthomyxovirus family.

The virus is transmitted by aerosol, wind, nose-to-nose contact, fomites such as tack, grooming equipment, machinery, feed and human contact. The virus is delicate within the environment and easily killed by heat, cold, desiccation and disinfectants.

The incubation period lasts 1–3 days and shedding of the virus may occur as early as 24 hours post-infection. Shedding may continue for 7–10 days.

This disease is uncommonly diagnosed in foals less than 8 months of age. Young immunologically inexperienced horses and unvaccinated horses are particularly susceptible to infection.

Clinical signs

- Signs include fever, lethargy and anorexia. Fevers may range from 39.1–41.7°C (102.5–107°F). These signs last between 1 to 5 days.
- Inflammation and irritation of the airways may cause affected horses to have paroxysmal coughing.
- Increased inspiratory and expiratory bronchovesicular sounds are often heard on auscultation of the chest.
- Serous and/or light mucoid discharge, muscle soreness and stiffness are also frequently noted in affected animals.
- Some animals may develop a secondary bacterial pneumonia because of decreased bacterial clearance by the mucociliary apparatus.

Diagnosis

- Influenza spreads rapidly and therefore rapid detection is necessary for control.
- Clinical diagnosis can be obtained with virus isolation from nasal or pharyngeal swabs, ELISA which detects viral nucleoproteins from nasopharyngeal swab samples and/or paired serology looking for a 4× rise in titers.

Treatment

- Treatment is mostly symptomatic.
- Non-steroidal anti-inflammatory drugs may be administered to resolve the pyrexia, and inappetence.
- Keeping the environment dust free with the soaking of hay, wetting down bedding and removing the patient from the barn when cleaning is imperative in preventing a secondary bacterial pneumonia.
- Broad-spectrum systemic antibiotics may be needed for animals suffering from secondary bacterial pneumonia.

Rhodoccocus equi pneumonia (Figs 5.52–5.67)

Pneumonia in foals, caused by *Rhodococcus equi* (*R. equi*) is a well-known worldwide problem. Other less common clinical manifestations of *R. equi* infections in foals include ulcerative enterocolitis, colonic/mesenteric lymphadenopathy, immune mediated synovitis and uveitis, osteomyelitis and septic arthritis.

Inhalation of contaminated dust particles is thought to be an important route for pneumonic infection of foals. Ingestion of the organisms is a significant route of exposure and immunization but may not lead to hematogenous pneumonia unless the foal has multiple exposures to very large numbers of bacteria.

Recent epidemiologic evidence indicates that foals that develop *R. equi* pneumonia are most commonly infected during the first few days

Figure 5.52: Immune mediated synovitis (tibiotarsal effusion) in a foal with *R. equi*. The foal was not lame at a walk or trot.

Figure 5.53: Uveitis in a foal suffering from *R. equi* infection. Note the green hue of the iris and aqueous humor which is characteristic of uveitis. Foals diagnosed with uveitis between the age of 3 to 6 months should have their lungs evaluated (i.e. ultrasound or radiographs) since this is a common extrapulmonary lesion with *R. equi* infection.

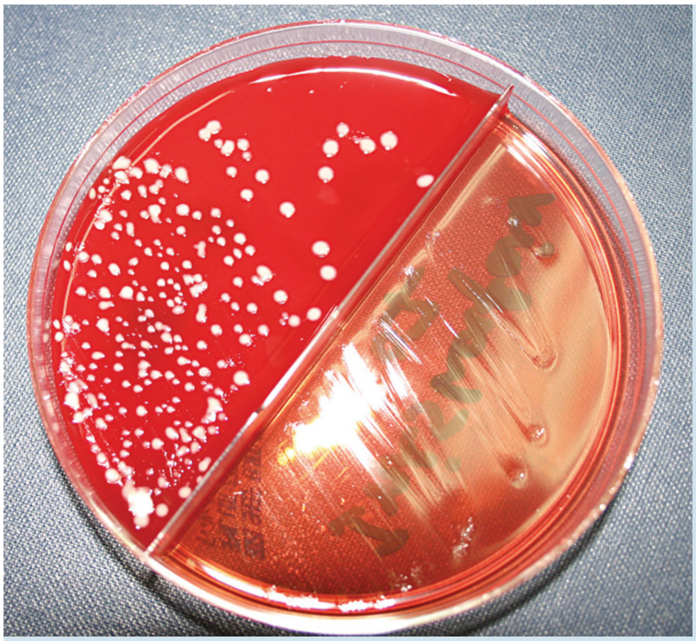


Figure 5.54: *R. equi*. colonies on a blood agar/McConkey plate after 48 hours of incubation. Note the raised, moist, translucent, and regular colonies on the blood agar and no growth on McConkey. Colonies are initially whitish in color but soon become a rose pink as in this image. This was a pure growth of *R. equi* obtained from a tracheal wash of the foal in Fig 5.61. In addition to the lesion imaged this foal had three other lesions of equal size.

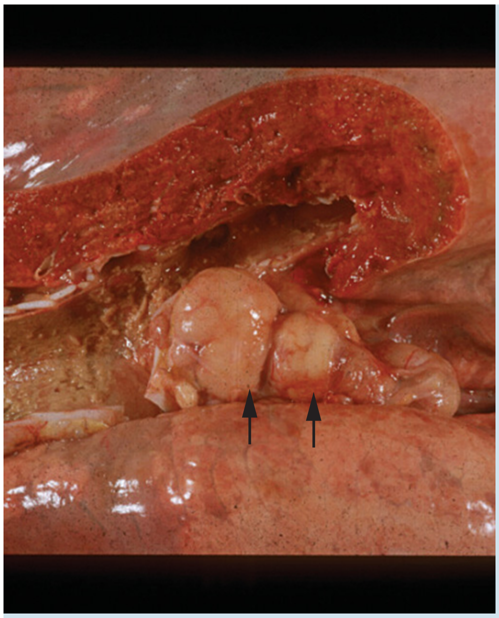


Figure 5.55: Hilar lymphadenopathy (arrows) secondary to *R. equi*.

Figure 5.56: Ultrasonographic examinations can be performed with a multifrequency 5.0- or 7.5-MHz linear transrectal transducer. The 7.5-MHz will be able to display a depth of 4–12 cm which is ideal for the thoracic examination of a foal. Isopropyl alcohol is copiously applied to the haircoat to provide surface contact between the transducer and the foal. The alcohol helps to reduce the intervening trapped air. The thorax was thoroughly scanned in a dorsal to ventral plane from the 16th to the 3rd intercostal space. Remember to assess the lung field deep to the triceps muscle as noted in this photo.

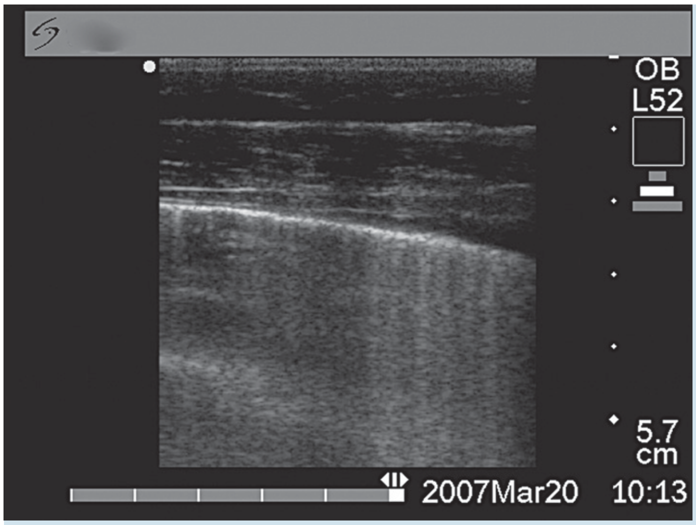


Figure 5.57: Comet tail artifacts noted in a healthy foal during a routine ultrasound examination. This is not a significant finding and would be categorized as a Grade 0.

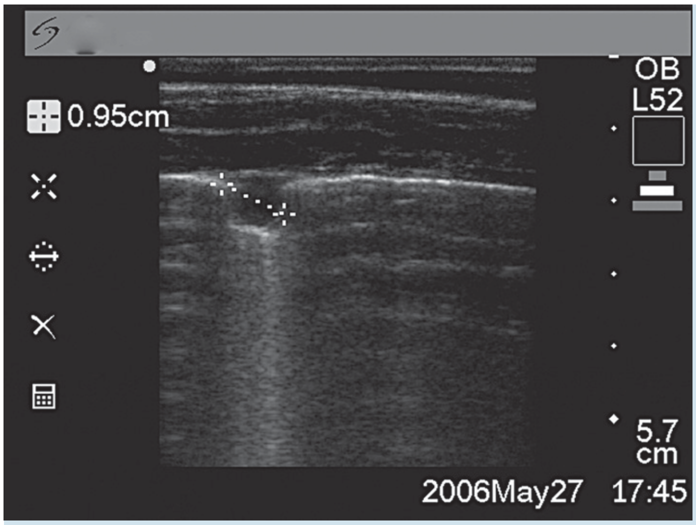


Figure 5.58: A Grade 1 lung consolidation. The consolidated region measures 0.95 cm in diameter.

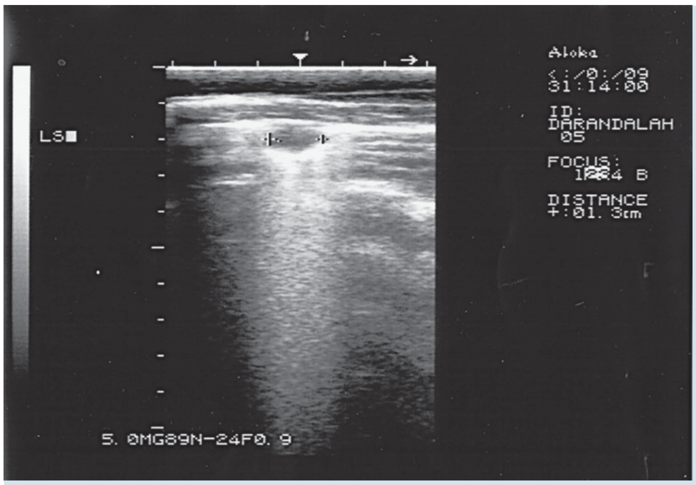


Figure 5.59: A Grade 2 lung consolidation. The consolidated region measures 1.3 cm.

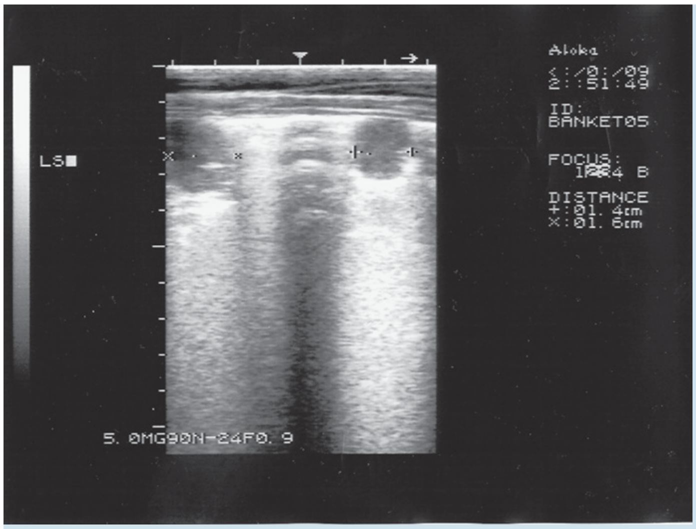


Figure 5.60: Two Grade 2 lesions imaged beside each other; the one on the left measures 1.6 cm and the one on the right 1.4 cm.

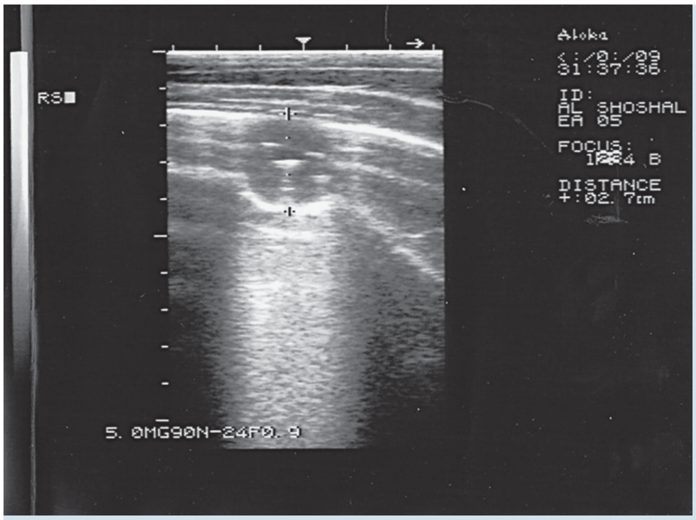


Figure 5.61: Grade 3 lung consolidation. The consolidated region measured 2.1 cm in diameter.

of life, but clinical signs do not develop until foals are 30–60 days of age or older.

The most common manifestation of *R. equi* in foals is a suppurative bronchopneumonia with extensive abscessation and suppurative lymphadenitis. The slow spread of the lung infection coupled with the remarkable ability of foals to compensate for the progressive loss of functional lung makes early diagnosis difficult.

Clinical signs

- Early clinical signs may only include a slight increase in respiratory rate and mild fever. These clinical signs are often missed, allowing the disease to progress.
- Therefore the respiratory signs are often apparently acute in onset. A smaller percentage of these foals may be found dead or more commonly present in acute respiratory distress with high fevers of 41°C (105–106°F) and no previous history of clinical respiratory disease.

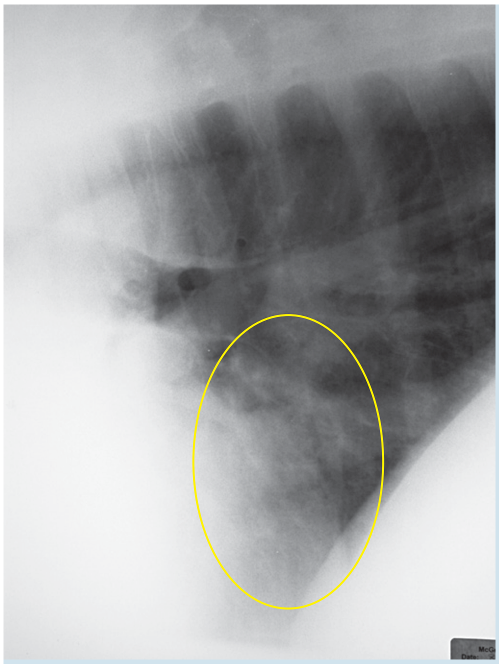


Figure 5.62: Thoracic radiograph of a grade 3 ultrasonographic lesion. The foal was previously diagnosed with a grade 6 ultrasonographic lesion (Figure 5.63) which was treated with azithromycin and rifampin. This radiograph was taken after 4 weeks of treatment. The thoracic radiograph of this foal is showing an alveolar pattern with ill-defined regional (yellow circle) consolidation in the caudoventral lung field just caudal to the cardiac silhouette. This radiograph is a prime example of how thoracic ultrasonography could delineate this foal's lesions before *significant* radiographic changes were evident.

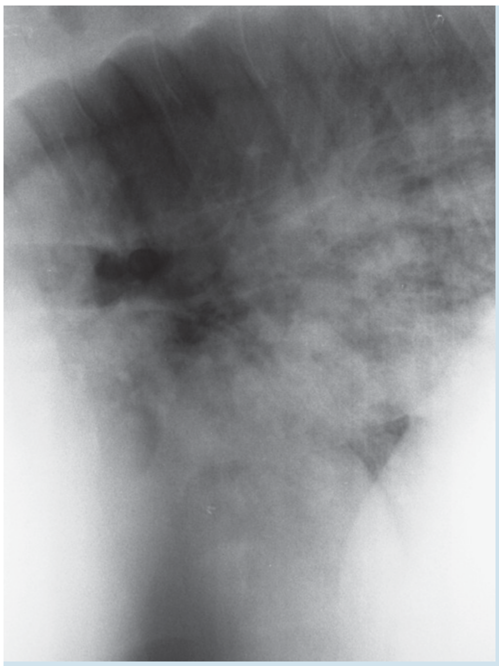


Figure 5.63: *R. equi* – standing lateral radiograph revealing multiple radiodense lesions throughout the lung. The animal was diagnosed with a grade 6 lesion on thoracic ultrasonography.

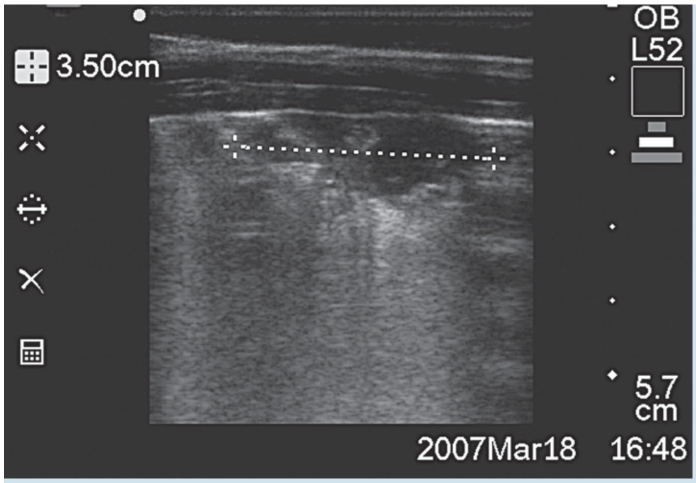


Figure 5.64: Grade 4 pulmonary abscess measuring 3.5 cm.

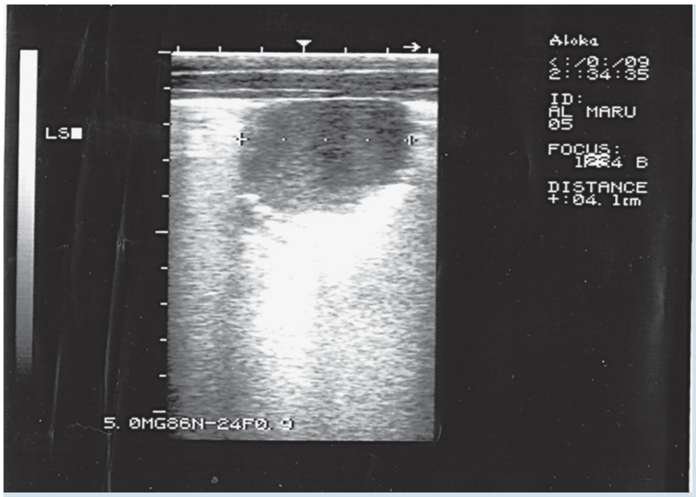


Figure 5.65: Grade 5 pulmonary abscess measuring 4.1 cm.

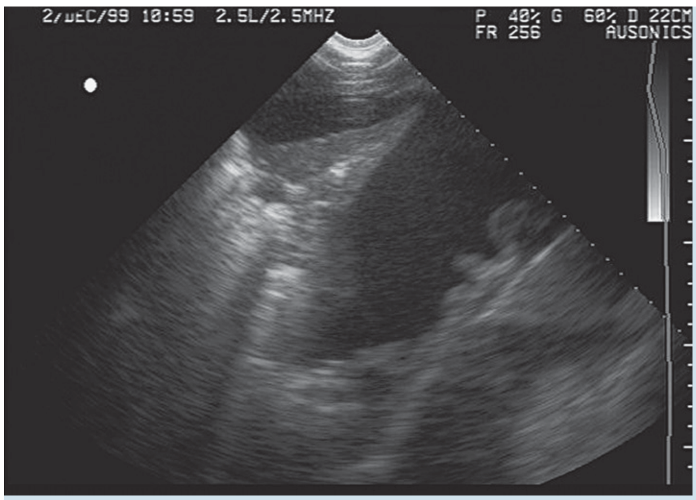


Figure 5.66: Grade 8 *R. equi* consolidation with pleural effusion.

Figure 5.67: Postmortem findings from a foal with severe *R. equi* infection. Multiple small abscesses and one large abscess can be seen.

- Up to 50% of foals with *R. equi* pneumonia have concurrent intestinal manifestations of disease with or without clinical signs (see Chapter 4, p. 112).
- Immune-mediated polysynovitis, particularly of the tibiotarsal and stifle joints, can be seen in 30% of cases with *R. equi* pneumonia.

Diagnosis

The insidious course of infection makes early diagnosis difficult. Recognition of foals with *R. equi* pneumonia prior to the development of clinical signs would likely reduce losses and limit costs associated with long-term treatment of affected foals.

Many diagnostic tests including complete blood cell count, fibrinogen level, thoracic ultrasound, radiographs and serology have all been used to help distinguish *R. equi* pneumonia from that caused by other pathogens. However, bacteriologic culture or PCR amplification combined with cytological examination of a tracheobronchial aspirate (TBA) are still the "gold standards" used to arrive at a definitive diagnosis.

A recent study suggested that serological assays, whether performed on single or paired samples, cannot be used to reliably establish, confirm or exclude a diagnosis of *R. equi* pneumonia in foals. These serological tests are problematic because of the widespread exposure of foals to this organism at a young age which would initiate appropriate antibody production.

Measurements of white blood cell count (WBC) or fibrinogen concentrations are non-specific indicators of infection or inflammation. A recent study revealed that although both measurement of fibrinogen concentrations and WBC concentration were useful for early identification of *R. equi*-infected foals, WBC was significantly better than measurement of fibrinogen concentrations. On the basis of their data on a farm where the prevalence of the disease is high, foals with a WBC concentration of <13,000 cells/μL are unlikely infected with a negative predicative value of 95.1% at a prevalence of 40%. Increasing the cut-off to >15,000 cells/μL greatly improves the positive predictive value. On an endemic farm results of a WBC >13,000 cells/μL or noting a foal with a fever would warrant a careful examination by the veterinarian. Foals with a WBC >14,000 cells/μL with no clinical signs of disease and normal lung sounds should be considered for additional diagnostic tests such as thoracic ultrasonography.

Ultrasonography may reveal abnormalities of the peripheral pulmonary parenchyma. If these abnormalities are detected then a TW and/or antibiotic treatment should be initiated. Farms with endemic *R. equi* that have suffered significant morbidity and/or mortality rates should be monitoring rectal temperatures 2× daily, with febrile foals selected for further testing (thoracic ultrasonography) or treatment. In the author's experience performing twice monthly thoracic ultrasonography (starting at 3 weeks of age) has demonstrated to be very effective for early recognition and reduction of mortality attributed to *R. equi* pneumonia on several endemic farms. The rationale for this early screening is the belief that earlier initiation of specific treatment will not only improve the prognosis for recovery but also reduce the treatment period (2 weeks rather than 4–8 weeks of antibiotic treatments). Thoracic ultrasonography can also be used to determine when the antibiotic therapy can be discontinued. Additionally, ongoing research suggests that thoracic ultrasonography may be a more sensitive indicator of subclinical or early clinical lesions than measurement of WBC.

Diagnostic thoracic ultrasonography has also been shown to be an accurate alternative imaging modality for detection of pulmonary pathology attributed to *R. equi* pneumonia in foals when thoracic radiography is not available.

A grading scale for lesions detected by ultrasonography, ranging from 0 (normal) to 10 (entire lung surface is affected) has been developed to aid in the documentation of lesions, assessment of treatment success and help with the communication and description of pneumonia. Pulmonary lesions are assigned a grade according to their severity. The foal's grade is determined not by the total number of lesions that are visualized but by the highest grade visualized. For example a foal with multiple grade 1 lesions of the left hemithorax as well as one grade 3 lesion would be identified as a grade 3 in the left thorax. This grading system is outlined below:

- GRADE 0: No evidence of pulmonary consolidation. Pleural irregularities which appear as vertical hyperechoic lines and are described as reverberation artifacts.
- GRADE 1: Lesions <1 cm in diameter/depth
- GRADE 2: 1.0–2.0 cm
- GRADE 3: 2.0–3.0 cm
- GRADE 4: 3.0–4.0 cm
- GRADE 5: 4.0–5.0 cm
- GRADE 6: 5.0–6.0 cm
- GRADE 7: 6.0–7.0 cm
- GRADE 8: 7.0–9.0 cm. If pleural effusion is present then the lesion is assigned this grade regardless of whether or not you have lesser grades of consolidation or abscessation
- GRADE 9: 9.0–11.0 cm
- GRADE 10: The entire lung lobe is affected.

Treatment

- Although controlled trials to evaluate optimal treatment are lacking, erythromycin and rifampin are considered standard treatment. This protocol is considered costly, labor intensive (3 times a day treatment) and can result in adverse reactions in treated foals (diarrhea and hyperthermia) and dams of foals on treatment (colitis).

- Other macrolides such as azithromycin (Zithromax®) and clarithromycin (Biaxin®) have been used for the treatment of *R. equi* in foals.
- The pharmacokinetics of azithromycin have shown that 10 mg/kg PO q24 h for the first 5 days of treatment and then every other day achieve appropriate MIC90 concentrations in bronchoalveolar cells and fluid. On the basis of pharmacokinetic values and minimum inhibitory concentrations of *R. equi* isolates, clarithromycin at a dose of 7.5 mg/kg PO q12 h can keep serum levels above the MIC90. Azithromycin and clarithromycin with rifampin can be used in combination for *R. equi* pneumonia that does not appear to respond to azithromycin alone. Foals that recover from *R. equi* pneumonia and make it to the race track have been shown to perform as well as expected.
- All macrolides may cause hyperthermia and foals should be stabled while on treatment. The cause of the hyperthermia is not exactly known but is believed to be related to exposure to sunlight rather than the ambient temperature. However, stabling in a cooled environment may aid foals in respiratory distress.
- Severe cases which present in respiratory distress, or foals which have a marked elevated respiratory rate, should have a blood gas analysis performed and may require oxygen insufflation.

Streptococcus equi var. *equi* (strangles) (Figs 5.68–5.73)

Streptococcus equi var. *equi* (strangles) is a Gram-positive bacterium.

Animals are exposed to *Strep. equi* from secretions of an affected horse via direct or indirect contact with a sick animal or an asymptomatic carrier. Infected horses shed the organism through the nasal passages so nose-to-nose contact of horses can spread the disease as well as coming in contact with secretions on fomites such as fences, water buckets or troughs, pitch forks, or even a human handler. Horse vans, stalls or public auctions are all possible sources for *Strep. equi*.

Figure 5.68: Swollen retropharyngeal region in a foal with *Strep. equi*. The foal presented with difficulty swallowing and respiratory stridor.

Clinical signs

- The initial clinical sign of strangles is a fever.
- Fevers usually occur 3–14 days after exposure.
- Affected animals usually start shedding the organisms several days after onset of fever. Abscessed lymph nodes occur shortly after the onset of nasal shedding.
- The retropharyngeal and submandibular lymph nodes are most commonly affected, but other lymph nodes of the head such as the parotid and cranial cervical are also commonly involved.
- Serum may ooze from the skin before the lymph nodes rupture and drain.

Figure 5.69: Feed material noted from the nares of the patient in Fig 5.68. On examination the pharyngeal region was collapsed and the animal was noted to be dysphagic.

Figure 5.70: Aspiration of a retropharyngeal lymph node. *Streptococcus equi* var. *equi* was cultured from the aspirate.

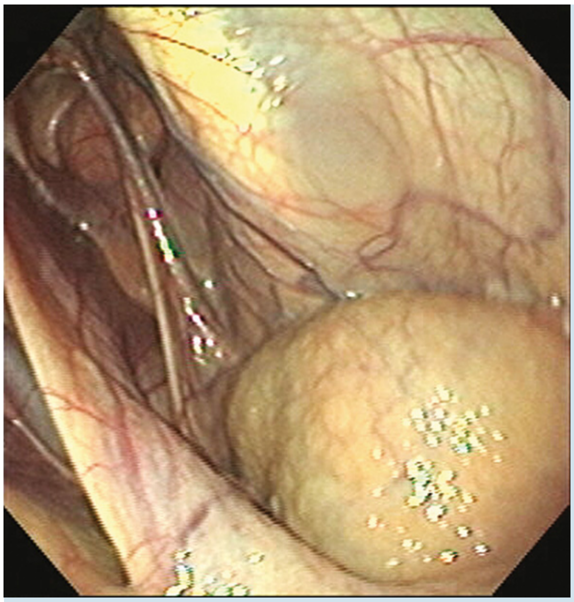


Figure 5.71: Enlarged lymph node on the ventral floor of the guttural pouch. The foal had a history of fevers and was tested for *Strep. equi* via a PCR pharyngeal flush. The flush was PCR positive.

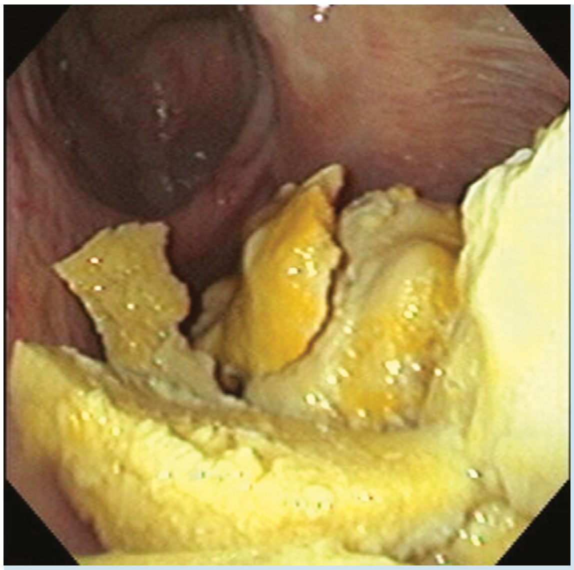


Figure 5.72: Chondroids in a guttural pouch of an animal diagnosed with *Strep. equi*. The animal had the guttural pouches lavaged with saline and infused with acetylcysteine to help dissolve the chondroids. Four treatments were necessary over a week for complete resolution.

- Affected animals may initially have a serous nasal discharge which progresses to mucopurulent and then purulent.
- Severely affected animals may present in respiratory distress from occlusion of the trachea caused by swelling of the retropharyngeal lymph nodes.
- Empyema of the guttural pouch may occur if the retropharyngeal lymph nodes rupture and drain internally into the pouches.
- The empyema may become inspissated and form chondroids, which are difficult to remove from the pouches.

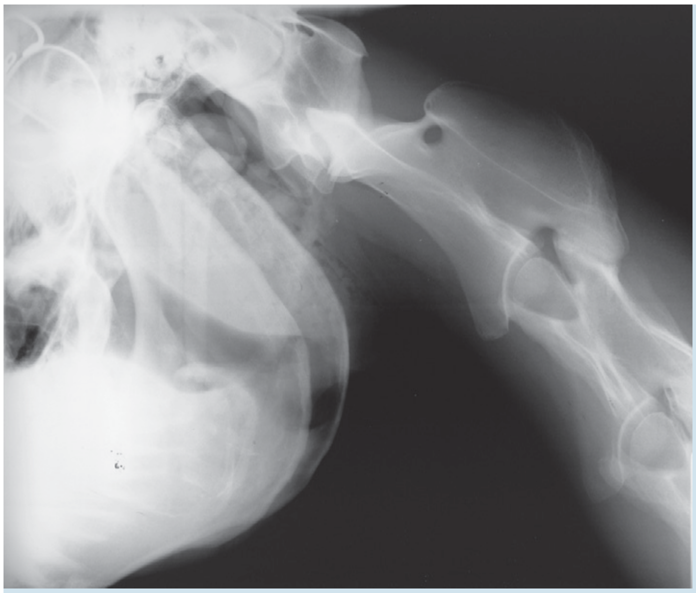


Figure 5.73: Radiograph of the skull demonstrating chondroids within the guttural pouches.

Diagnosis

- A presumptive diagnosis can be based on clinical signs of the characteristic abscesses. The confirming diagnosis via culture can be obtained from a draining abscess, a nasal swab, a pharyngeal wash, or a guttural pouch wash.
- A polymerase chain reaction (PCR) can also be used as a diagnostic tool. PCR detects the presence of DNA and does not distinguish between live or dead samples. PCR is considered more sensitive than culture, but may also yield false negatives.
- Serology is available to aid in diagnosis. An ELISA test measures *Strep. equi* specific M-protein, but does not distinguish between infected and vaccinated animals.

Asymptomatic carriers may be present on the farm and spread *Strep. equi* to other horses.

- The use of pharyngeal washes and PCR analysis can be performed on all new arrivals to the farm, or horses exposed to *Strep. equi*, before commingling. The following items are needed for the wash:
  - Lip chain (or sedation): disinfect after use with chlorohexidine (soak for 10 minutes)
  - Saline
  - 60 cc syringe
  - Gloves
  - Plastic cups
  - Polypropylene catheter 56 cm/22 in. 8 French and 5 French for foals:
    - this tube is passed into the nostril and lodged into the pharynx
  - Collection tubes.
- The animal has the pharynx flushed with the 60 cc of saline, and the exudate running out of both nostrils is collected for PCR analysis.
- Positive test:
  - Isolate the animal(s)

- ♦ The guttural pouches should be scoped/radiographed for assessment of chondroids (see Figures 5.72 and 5.73)
- ♦ Guttural pouches are flushed with saline and a topical gelatin-based sodium penicillin gel is infused into each pouch
- ♦ Treat with systemic procaine penicillin (22,000 IU/kg IM BID) or ceftiofur 2.2 mg/kg IV or IM SID) for 1 week followed by 2 weeks of TMS (30 mg/kg PO BID) if sensitivity testing indicates efficacy.
- ♦ Re-evaluate the pharyngeal wash in 4 weeks via PCR.

Treatment

- Foals with clinically **suspected** *Strep. equi* should be isolated immediately.
- Most foals affected with *Strep. equi* do not require treatment or antibiotics therefore allowing maturation and eventual rupture/drainage of the abscess.
- Foals exhibiting additional clinical signs such as profound fever, depression, anorexia or dyspnea require antibiotic treatment. Penicillin is the antibiotic of choice for strangles. Trimethoprim sulfa has mixed reports on its efficacy against *Strep. equi*. Rifampin and the macrolides (azithromycin or clarithromycin) have an excellent spectrum of activity against *Streptococcus* infections and can be used in cases in which owner compliance may be questionable.
- Foals in respiratory distress from abscesses compressing the trachea require a tracheostomy below the affected site.
- Non-steroidal anti-inflammatory medications (phenylbutazone, flunixin meglumine, or ketoprofen) may also aid in alleviating fever and improving anorexia.
- Animals affected with guttural pouch empyema should be placed on systemic antibiotics and have their guttural pouches flushed and infused with a penicillin gel.

Streptococcus equi var. *zooepidemicus* lymphadenopathy

Unlike *Streptococcus equi* var. *equi* this bacterium is considered a secondary pathogen. *Streptococcus equi* var. *zooepidemicus* usually affects horses that have either suffered an upper respiratory infection, e.g. herpes virus infection, or are immunocompromised. It is not considered a primary pathogen like *Streptococcus equi* var. *equi*.

Clinical signs

Clinical signs are almost identical to *Streptococcus equi* var. *equi*.

Diagnosis

Cultures are needed to confirm a diagnosis of *Streptococcus equi* var. *zooepidemicus*.

Treatment

- Enlarged or painful abscessed lymph nodes can be lanced and drained:
  - ♦ flush with saline and infuse penicillin.
- Foals exhibiting additional clinical signs such as profound fever, depression, anorexia or dyspnea require antibiotic treatment. Penicillin is the antibiotic of choice. Trimethoprim sulfa has mixed reports on its efficacy against *Strep. zooepidemicus*. Rifampin and the macrolides (azithromycin or clarithromycin) have an excellent spectrum of activity against *Streptococcus* infections but may cause antibiotic induced diarrhea in older foals.
- Foals in respiratory distress from abscesses compressing the trachea require a tracheostomy below the affected site.
- Non-steroidal anti-inflammatory medications (phenylbutazone, flunixin meglumine, or ketoprofen) may also aid in alleviating fever and improving anorexia.

Pneumocystis carinii (Fig 5.74)

Formerly considered a protozoan organism, *P. carinii* has recently been reclassified as a fungus. The infective stage or source of *P. carinii* is unknown but recent investigations suggest that it may be transmitted in water or by an air-borne route. *P. carinii* pneumonia is thought to occur primarily in immunocompromised foals as a complication of some other serious illness such as infectious pneumonia or severe combined immunodeficiency (SCID).

Clinical signs

- Dyspnea occurs secondary to the plasmacytic lymphocytic interstitial pneumonia and the flooding of the alveoli with foamy exudate.
- It is usually an acutely fatal disease.

Diagnosis

- It is very difficult to obtain a diagnosis ante mortem.
- The use of thoracic radiographs may show a reticulonodular pattern (miliary).
- The use of either a bronchoalveolar lavage or a transtracheal wash has been suggested for identifying these organisms intracellularly in macrophages but results may be variable. Confirmation is by identification of the organisms by either silver staining or immunostaining.

Treatment

- Prognosis is considered guarded but successful treatment has been achieved with the use of either potentiated sulfonamides and/or

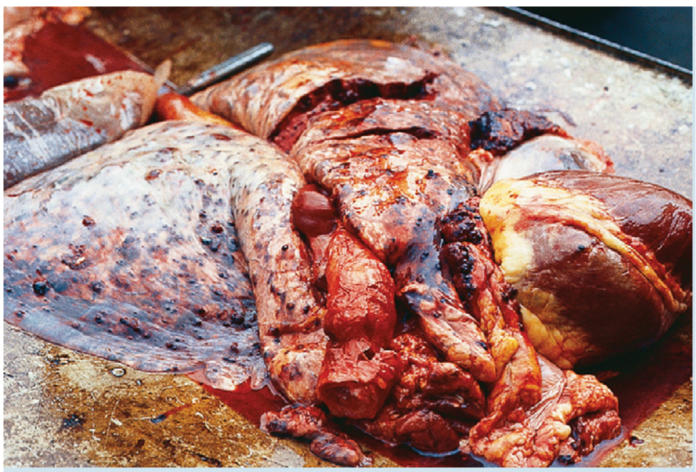


Figure 5.74: Pneumocystis – postmortem lung (from Knottenbelt DC et al 2004 Equine neonatal medicine and surgery. Saunders, Philadelphia).

dapsone 3 mg/kg (1.4 mg/lb), PO, q 24 h (dose extrapolated from human data). Dapsone is an antibiotic used to treat *P. carinii* infections in humans who cannot be administered sulfa-containing medications. Dapsone may be a useful adjunct to traditional treatment for *P. carinii* pneumonia in horses or as a sole medication for horses that cannot tolerate other treatments.
- Treatment is usually prolonged (45–50 days).

Pneumothorax (Figs 5.75 & 5.76)

Pneumothorax occurs when air enters the pleural space and reduces the negative pressure that keeps the lungs expanded and allows the horse to breathe.

Pneumothorax is characterized as either open or closed. An open pneumothorax results in an injury to the thoracic wall leading to an influx of free air into the pleural space and lung collapse. A closed pneumothorax is the leakage of air into the pleural space from a pulmonary source such as a bronchopleural fistula. A tension pneumothorax occurs when a section of traumatized lung acts like a "valve" that allows the air to enter but not leave the pleural space.

Clinical signs

- Foals usually present in respiratory distress with exaggerated abdominal lift, elevated respiratory rate and flared nostrils.
- Bilateral pneumothorax will result in cyanosis and severe dyspnea.

Diagnosis

- Thoracic radiography reveals a horizontal shadow beneath the thoracic transverse processes, which is consistent with a "line" representing the collapsed lung(s).
- Thoracic ultrasonography will reveal horizontal air artifacts in the midthoracic or dorsal regions thereby not allowing the examiner to identify the sliding motion of the visceral pleura against the parietal pleura.
- If radiological diagnostics are not available then the clinician can use suction via a thoracocentesis as a diagnosis.

Treatment

- The treatment of choice is **prompt** removal of free air via a thoracocentesis and suction. This procedure rapidly re-expands the lung and relieves the respiratory distress.
- If an open pneumothorax is diagnosed then surgical closure is warranted.

Fractured ribs (Figs 5.77–5.89)

Thoracic trauma was described in newborn foals over 70 years ago. In a field prospective study of this condition, Jean et al reported that 20% of foals less than age 3 days on a large Thoroughbred stud farm had evidence of thoracic trauma, which included either rib fracture or costochondral dislocation, but no clinical problem associated with the condition was identified in this 6-month study.

In foals presented to a private intensive care unit, rib fractures significantly contributed to morbidity and mortality in affected neonates. In a retrospective study on the pathological features of thoracic trauma, Schambourg et al (2003) reported that in 19 of 67 examined foals, rib fractures were considered the cause of death. The additional manipulation of hospitalized patients may be a factor in the increased mortality rate of this group when compared with foals that have no additional abnormalities or illnesses.

Clinical signs

- Signs that should direct an examiner's attention to the possibility of costal fractures include groaning or grunting in the foal especially during palpation examination of the thoracic wall, respiratory

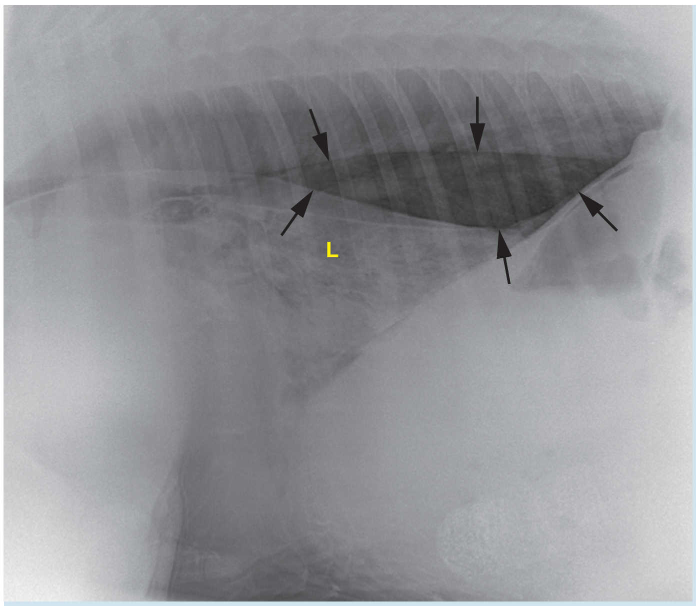


Figure 5.75: Open pneumothorax in a 2-day-old foal that ran into a post in the field. Note the free air (solid arrows) evident in the thorax above the collapsed lung (L). The laceration was sutured and then a thoracocentesis was performed to evacuate (via suction) the air in the thoracic cavity.

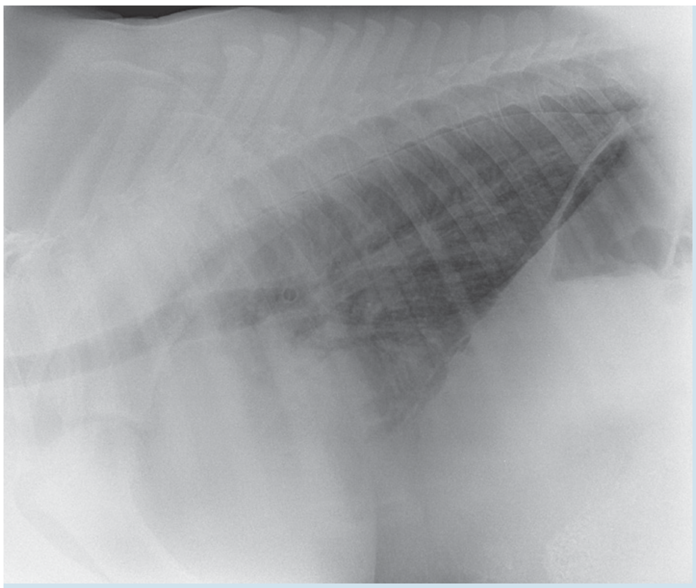


Figure 5.76: Resolution of the pneumothorax in Fig 5.75.

Figure 5.77: Flail chest in a 1-week-old foal with fractured ribs. Note the left chest wall sinks inward during inspiration (arrow).

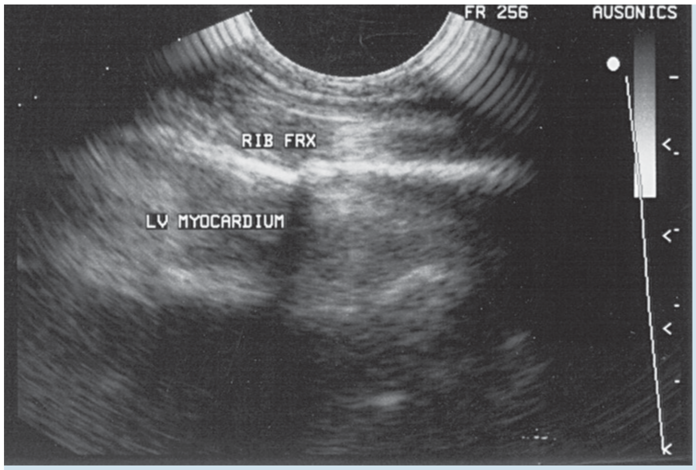


Figure 5.78: Ultrasonographic examination of the rib revealed a minimally displaced rib fracture over the heart. This foal was treated conservatively with 35 days of stall rest. Rib fractures should be re-assessed prior to turnout.

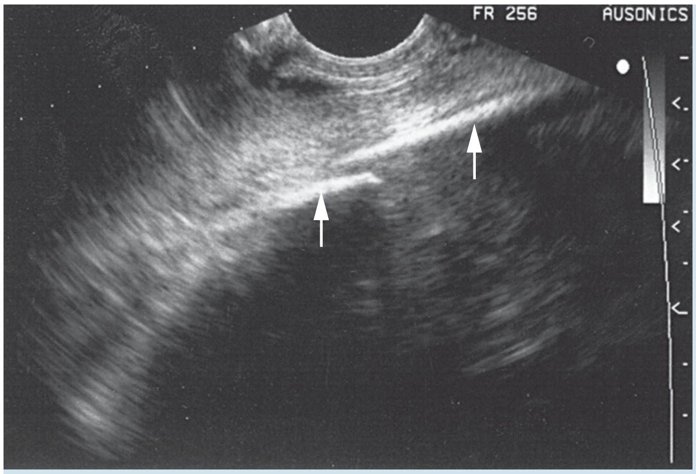


Figure 5.79: Ultrasonographic examination of the thorax revealing a mildly displaced rib fracture. The rib is the hyperechoic line (arrows).

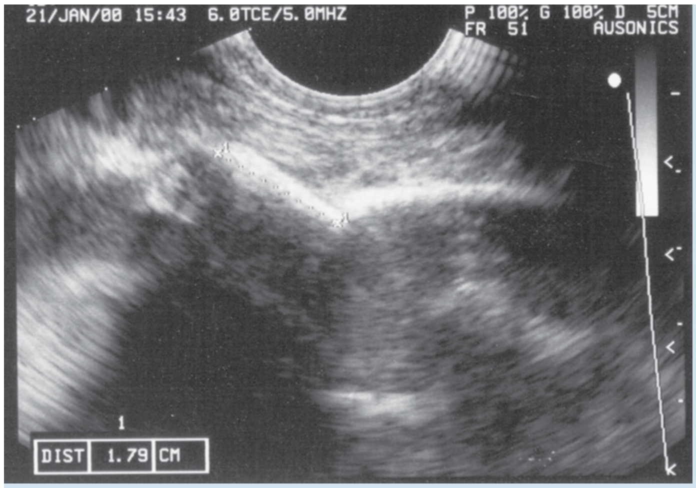


Figure 5.80: Measuring the distal fragment of a fractured rib. The distal fragment should be ideally >1.5 cm for an internal fixator to be utilized.

Figure 5.81: Gross pathology illustrating distal fragments of several fractured ribs displaced medially. This foal was a severe dystocia in which manual assistance was necessary to deliver the foal. The foal died secondary to cardiac arrest from a punctured heart by one of the fragments (see Fig 5.82).

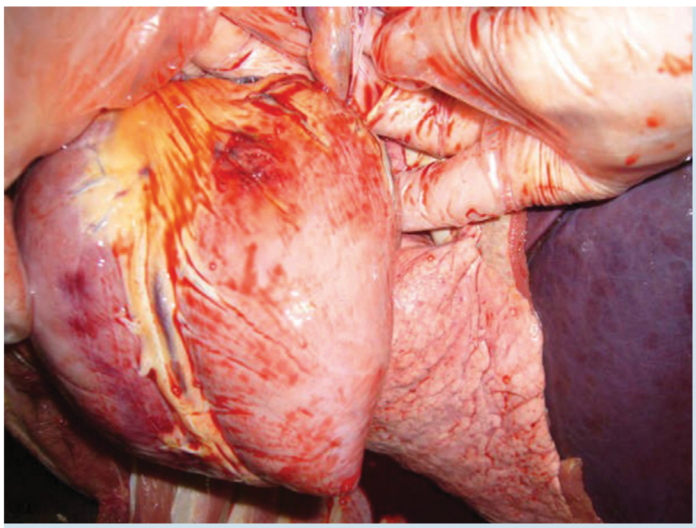


Figure 5.82: Laceration and bruising of the myocardium caused by multiple rib fractures, which resulted in the death of this 2-day old foal.

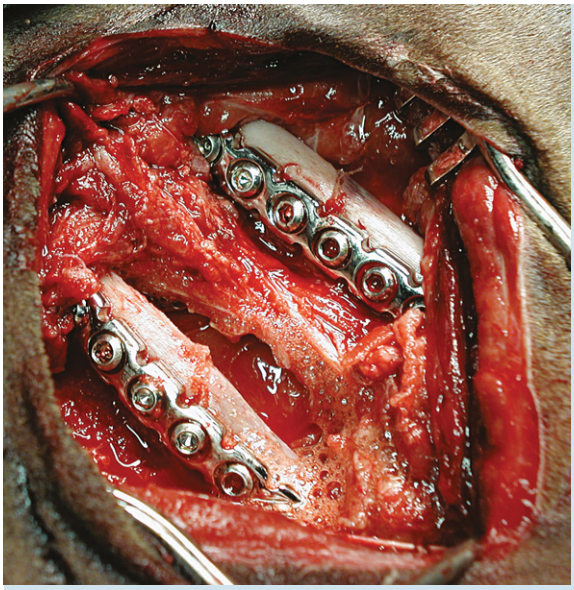


Figure 5.83: Open reduction/internal fixation technique for stabilization of fractured ribs. Bone forceps or towel clamps are utilized to grasp the proximal and distal fracture ends and distract the over-riding fracture ends with traction. A 2.7 mm reconstruction plate (4–8 hole) is contoured and secured to the rib with self tapping cortical screws. Orthopedic wire is placed around the rib and plate above and below the fracture line to offer further stability.

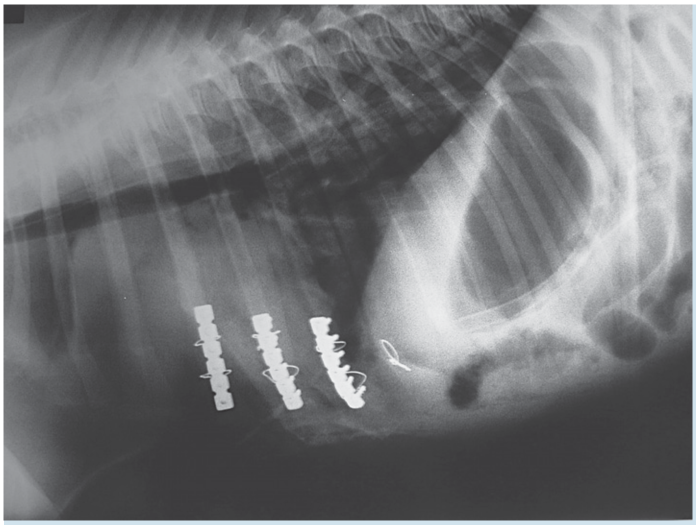


Figure 5.84: Thoracic radiograph of a foal with fractured ribs adjacent to the heart that were stabilized with three reconstruction plates. These plates will not be removed after the fracture has healed.

distress, plaques of subcutaneous edema overlying the ribs or along the ventrum of the thorax, especially behind the elbows, and flinching when the rib area is palpated.

- Fractured ribs are attended by significant pain in humans, but after several days of hospitalization many foals will demonstrate normal vigor and play activity in the stall, even when multiple broken ribs are present. Analgesic administration should be judicious in affected foals, as any movement or jostling of the thorax can cause rib fragments to lacerate internal vascular structures or the heart, resulting in sudden death. Not surprisingly, myocardial laceration or puncture are unequivocally fatal events.

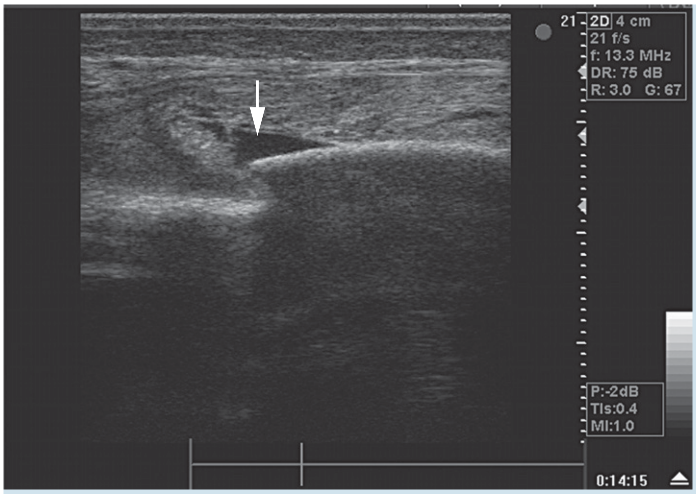


Figure 5.85: Ultrasonographic image of moderately displaced rib fracture at 2 days; note the hematoma at the site (arrow).

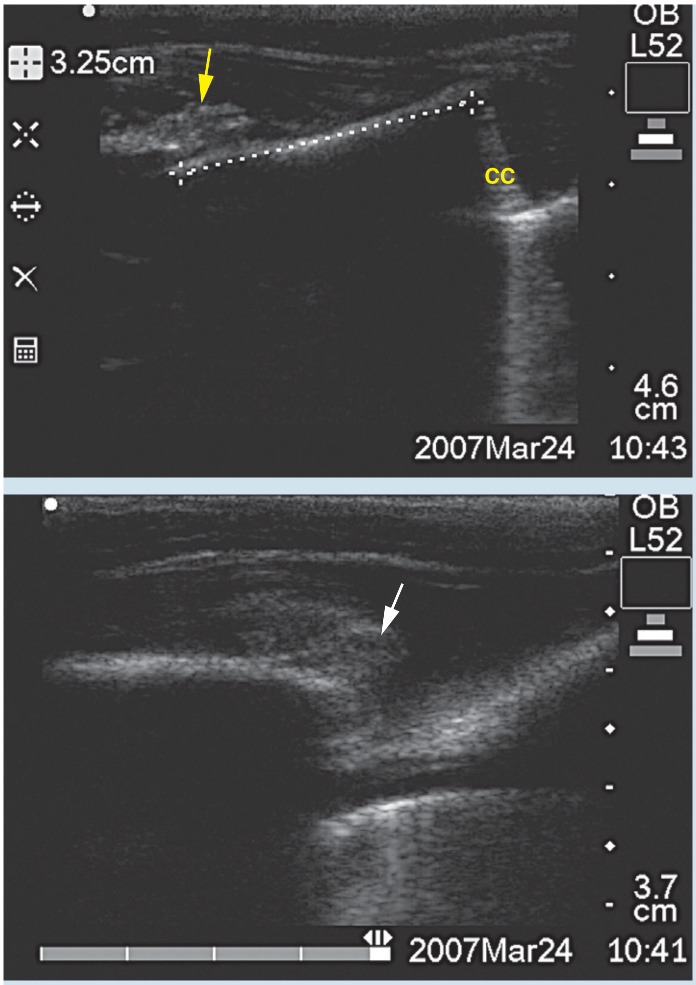


Figure 5.86: Ultrasonographic images of healing rib fractures at 8 days. Note the callus formation at the fracture sites (arrows) and the costochondral junction in the upper image (cc). These images were taken from a foal that sustained multiple rib fractures ranging from mildly to severely displaced, during an assisted posterior presentation delivery.

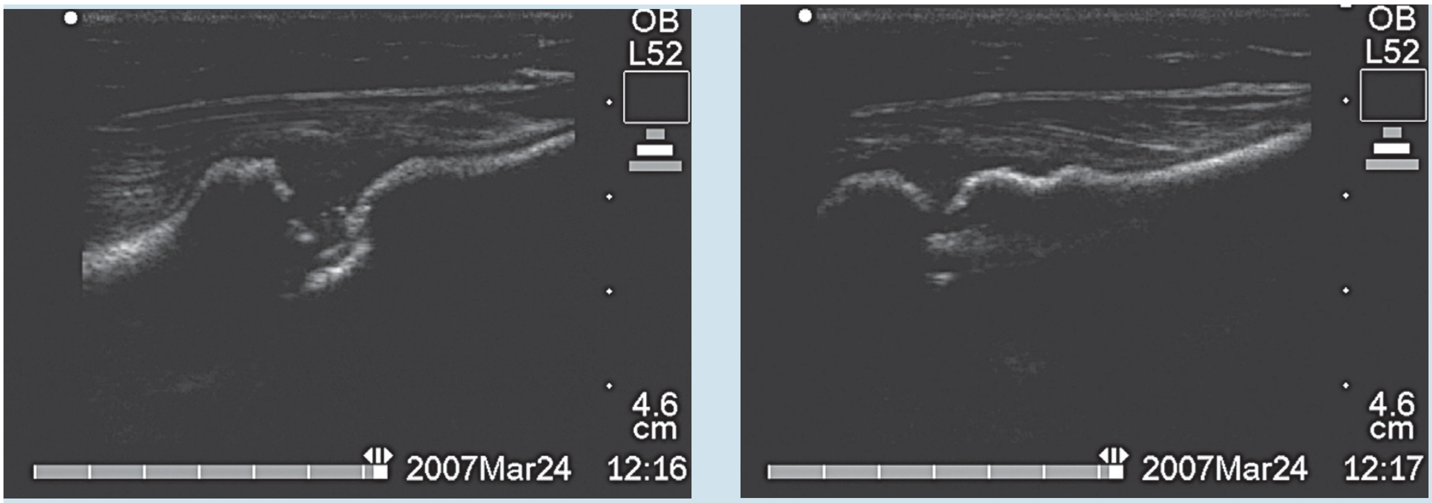


Figure 5.87: Ultrasonographic images of healing rib fractures at 4 weeks post-fracture. The image on the left is from the same foal as Fig 5.86. The image on the right is from a foal that was not known to have sustained a rib fracture at birth and was found incidentally during routine thoracic ultrasound as part of an *R. equi* detection program. Note the smaller callus in the image on the right from what was probably a mildly displaced fracture compared to the large callus in the image on the left from a severely displaced fracture.

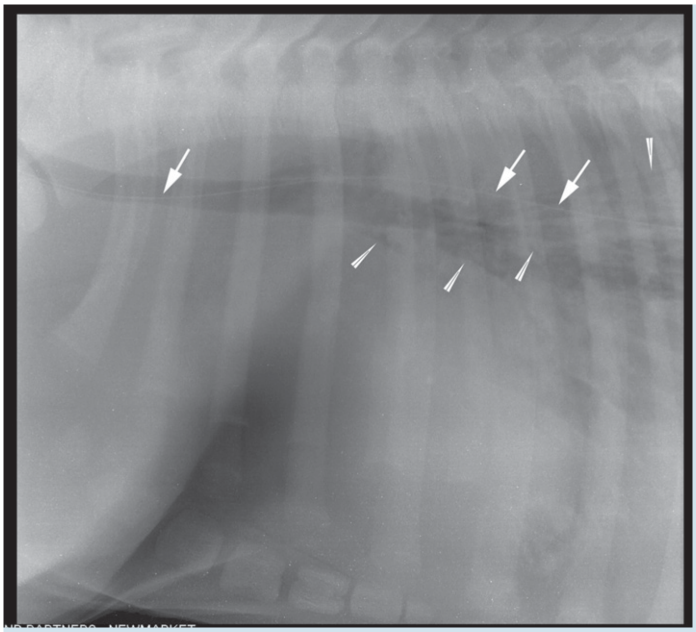


Figure 5.88: A right lateral thoracic radiograph from a 2-day-old Thoroughbred foal with fractures of the right fourth, fifth and sixth ribs. In this view it is difficult to discern the fractures but there is a generalized increase in opacity in the ventral thorax due to haemothorax. In the dorsal lung field there is a diffuse interstitial pattern and several air bronchograms (arrowheads) due to atelectasis and concurrent pneumonia. Note an indwelling radiopaque nasogastric feeding-tube is visible (arrows). Same foal as Fig 5.89.

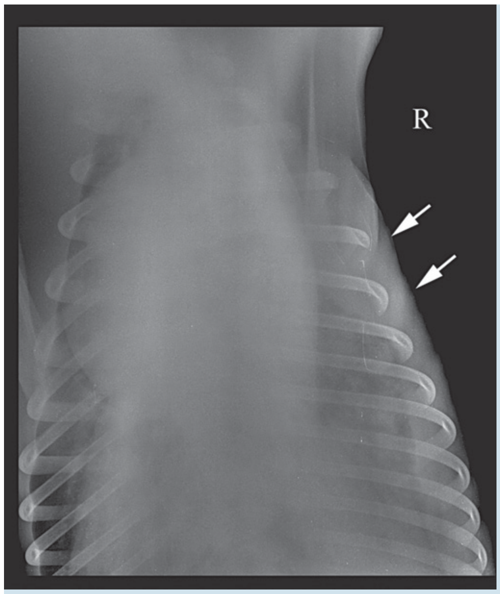


Figure 5.89: A dorsoventral thoracic radiograph from a 2-day-old Thoroughbred foal with fractures of the right fourth, fifth and sixth ribs. The fifth and sixth ribs are displaced medially (arrows). Same foal as Fig 5.88.

- Flail chest occurs when several consecutive ribs are fractured, leading to an incompetent segment of chest wall. The foal's respiratory efforts are hampered by the failure of the affected rib arcade to lift and participate in the process of creating negative pleural pressure for inspiration. During expiration, the failure of the affected chest segment to collapse as a unit likewise impedes the normal development of positive airway pressure and timely exhalation. When a flail chest is present, the involved segment of chest wall will sink inwards during the inspiratory excursions of the abdomen and diaphragm.

Diagnosis

- Palpation of the thoracic wall should be part of any routine neonatal examination. Audible or palpable crepitation or a clicking sensation when the hand is gently pressed over an affected area is common. Not all fractured ribs can be detected by palpation especially those that are only mildly displaced.
- The fractured ribs themselves can be well-visualized with ultrasonography, even when non-displaced, and the technique requires less positioning and manipulation of the injured patient than does radiography.
- The degree of fracture segment displacement and the proximity of bone ends to the heart can be immediately visualized, and the presence of pericardial effusion and peritoneal fluid are also more

easily and sensitively evaluated with ultrasonography than with radiography.

- The use of ultrasonography permits the examiner to identify even small volumes of pleural fluid, to immediately ascertain whether fluid in the chest is unilateral or bilateral, to characterize it as cellular and therefore likely to be blood in neonatal foals, and to observe changes associated with contusion and/or pneumonia in the underlying lung.
  - An undisplaced fracture is defined as a break in the margin without displacement.
  - A mildly displaced fracture is defined as a break in the margin with displacement not more than 1 mm (corresponding to the width of the anterior echogenic line).
  - A moderately displaced fracture is defined as a break in the cortex with displacement of more than 1 mm but less than 4 mm.
  - Severely displaced fractures are defined by displacement of more than 4 mm.

Treatment

The long-term monitoring of foals is done with serial ultrasonographic imaging:

- The formation of a hematoma or thrombus at the broken bone ends is typical of a rib fracture injury in the acute stages.
- Serial examinations document the evolution of this finding to bony callus formation, and eventual smoothing and remodeling of the callus with time.
- Four to six weeks are considered to be necessary for stabilization of the thoracic wall following rib fractures.
- Deformation of the lung surface and thickening of the visceral pleura may be semi-permanent to permanent findings in the long-term assessment of these foals. This is important to consider if routine thoracic ultrasonography is used as part of an *R. equi* detection program.

Foals that have severely displaced fractures that are adjacent to the heart or causing significant pulmonary contusions are considered surgical candidates:

- There are two basic techniques employed for repair of fractured ribs; one involves open reduction/internal fixation, and the other closed transcutaneous repair with an external stent. The use of the open reduction/internal fixation has been used on the vast majority of the foals at the author's clinic and has a higher surgical success rate than other techniques.

Diaphragmatic hernia (Figs 5.90–5.92)

Diaphragmatic hernias are categorized as either congenital or acquired. Congenital diaphragmatic hernia can occur in neonates when closure of the crura does not occur. Blunt trauma such as fractured ribs, falls, kicks or a dystocia can cause tearing or rupture of the diaphragm resulting in an acquired diaphragmatic hernia.

Clinical signs

Unlike adults with diaphragmatic hernias that frequently show no clinical signs, affected foals may present with colic, exaggerated respiratory pattern and toxic shock secondary to bowel incarceration or strangulation.

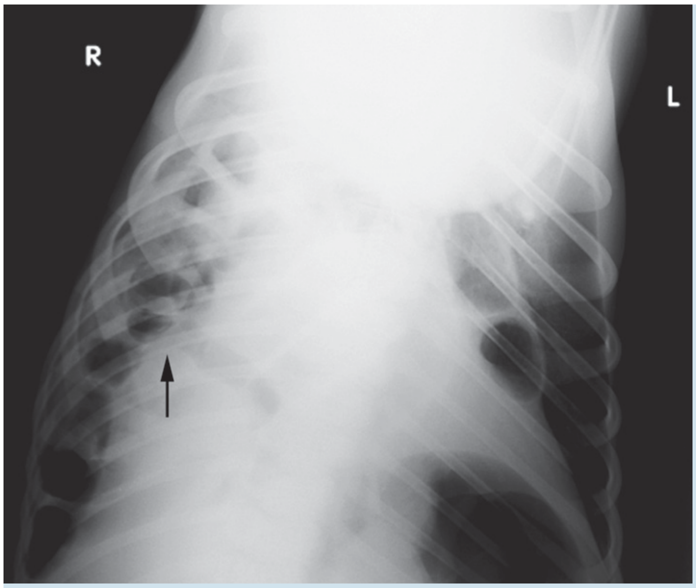


Figure 5.90: Dorso-ventral radiograph of a foal with a diaphragmatic hernia revealing gas-filled loops of bowel with in the pleural cavity (arrow).

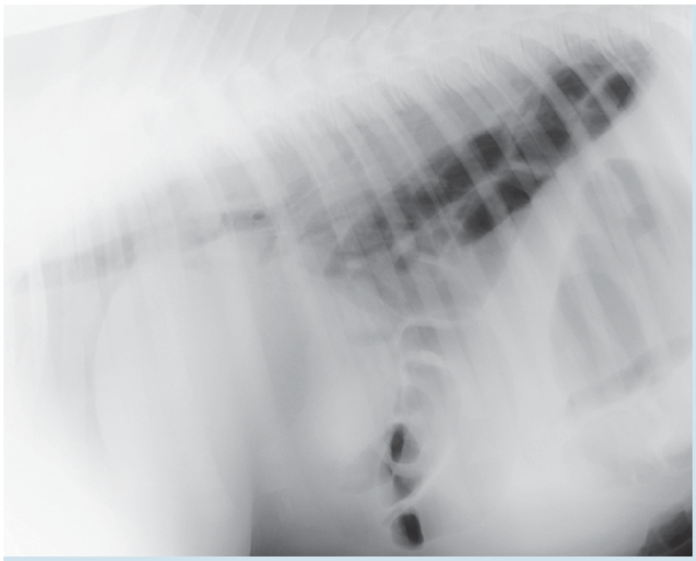


Figure 5.91: Lateral radiograph of the same foal again demonstrating gas-filled loops of bowel within the thoracic cavity.

Diagnosis

- Auscultation of the chest may reveal absence of normal bronchovesicular sounds or even reveal borborygmi.
- Thoracic radiographs will reveal gas-filled loops of bowel within the pleural cavity. Ultrasound of the thorax will reveal abdominal viscera in direct contact with the heart and lungs.

Treatment

- Surgical repair by either primary closure or the use of a surgical mesh.
- The animal should receive mineral oil by nasogastric tube and/or enemas to help loosen its stool following surgery. By loosening the

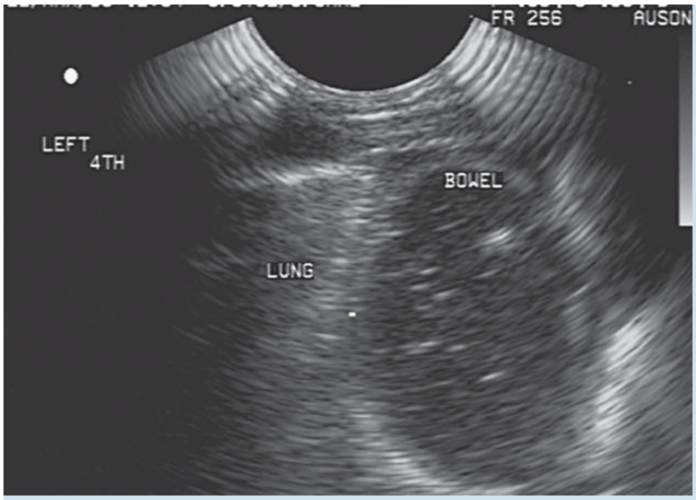


Figure 5.92: Ultrasonographic image of the thorax of a foal with a diaphragmatic hernia revealing distended bowel in the thoracic cavity. Note the distended colon adjacent to the lung. This foal was in marked respiratory distress. Surgical correction was achieved.

stool, the animal will decrease the amount of pressure exerted on the diaphragm when defecating.

Tracheal perforation (Figs 5.93–5.95)

Tracheal perforation secondary to blunt trauma is an uncommon occurrence noted in foals.

Clinical signs

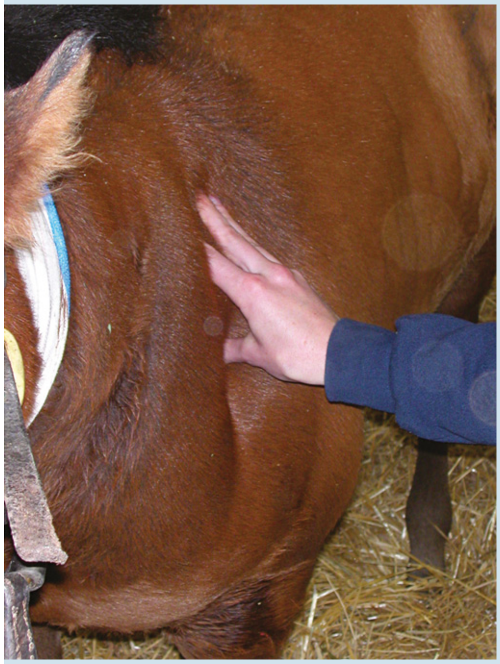
- Subcutaneous emphysema
- Stridor
- Bilateral hemorrhagic nasal discharge
- Tachypnea secondary to pneumomediastinum
- Endotracheal bleeding and mild colic signs.

Diagnosis

- Tracheoscopy is needed for confirmation of the tracheal laceration.
- Thoracic radiography can aid in the diagnosis of secondary pneumomediastinum.

Treatment

- Small tears may form a fibrin seal rapidly within 24–48 hours.
- Larger tears should be treated promptly to avoid infection, obstruction from peritracheal tissue and progression of subcutaneous emphysema to pneumomediastinum with resultant pneumothorax. A temporary tracheostomy distal to the site of tracheal perforation may have to be performed in such cases. This procedure diverts air away from the site of tracheal injury, helping restore airway control by decreasing the subcutaneous emphysema and aiding in the resolution of the injury.
- Complications associated with intratracheal granulation tissue at the site of tracheal trauma may respond to laser ablation therapy.



Figures 5.93: Subcutaneous emphysema in a foal that was kicked by another mare in the neck and sustained a tracheal perforation.

Bacterial pneumonia (Figs 5.96–5.105)

Pneumonia is the leading cause of morbidity and mortality in foals. Pneumonia in neonates is often associated with septicemia but may also occur secondary to meconium or milk aspiration.

The etiology of foal pneumonia is complex because of the large number of factors that foals are exposed to which can set the animal up for a bacterial pneumonia. The majority of older foals develop bacterial pneumonia following a viral infection. Viral agents damage

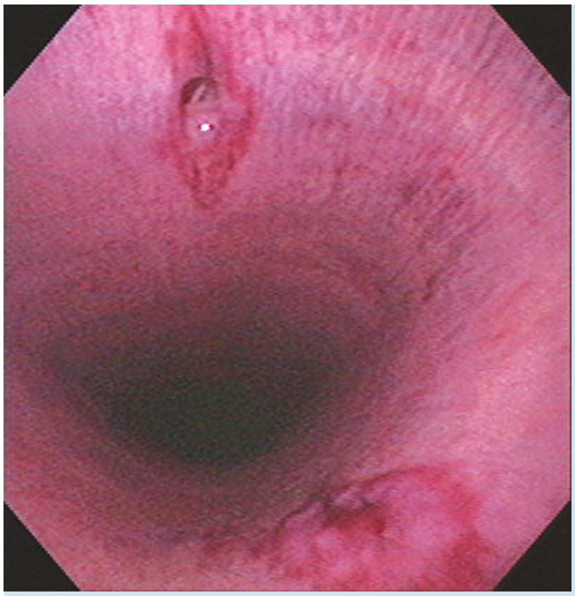


Figure 5.94: Cervical tracheoscopy demonstrated a 1 cm longitudinal perforation of the dorsal tracheal membrane of the proximal cervical trachea with associated mucosal hyperemia and edema ventrally.

Figure 5.95: A temporary tracheostomy distal to the site of tracheal perforation. This image is of a weanling with a recent tracheal perforation. Note the subcutaneous emphysema is not as apparent in the early stages.

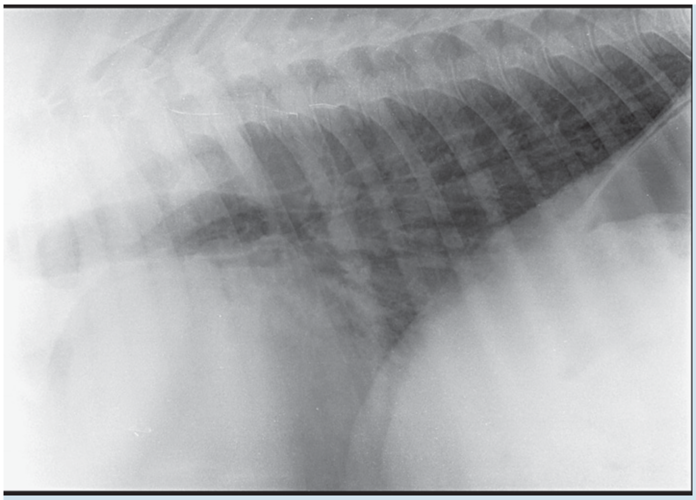


Figure 5.96: A lateral thoracic radiograph from a 6-week-old Suffolk Punch foal with bronchopneumonia – there is a generalized bronchointerstitial pattern.

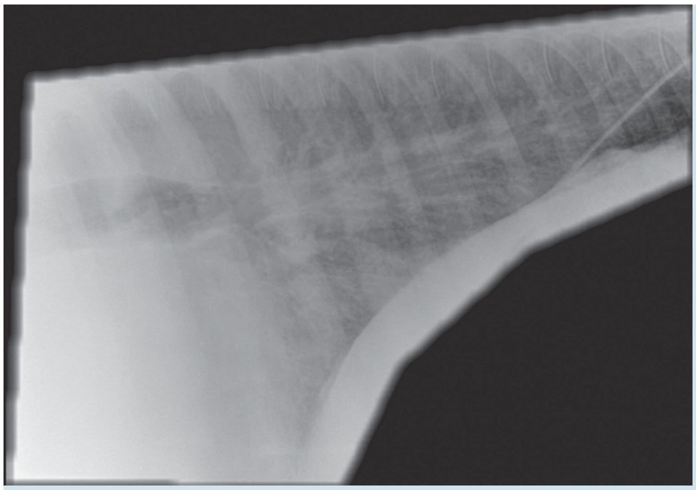


Figure 5.97: A lateral thoracic radiograph from an 11 week-old Suffolk Punch foal with bronchopneumonia: There is a marked generalized bronchointerstitial pattern. Same foal as Fig 5.96.

epithelial cells in the respiratory tree resulting in desquamation and focal erosion of the respiratory epithelium, interruption of the protective mucociliary blanket, and impairment of clearance mechanisms.

Parasites can also predispose to bacterial pneumonia by causing immunosuppression secondary to unthriftnesses or the migration of ascarids through the lung.

Clinical signs

- Clinical signs vary considerably with some foals having high respiratory rates while others may only have an occasional cough.
- Coughing, either intermittent or paroxysmal, is an important early indicator for respiratory disease.
- Respiratory rates greater than 30 BPM warrant further diagnostic evaluation which should include auscultation of the chest to assess if inspiratory or expiratory crackles or wheezes are noted.
- Most severely affected foals will have flared nostrils and an abdominal component to breathing with minimal thoracic excursion.
- Bilateral nasal discharge from profuse to scant and fevers are common findings in foals.

Diagnosis

Careful anscultatory examination of the chest (see p. 132) may reveal adventitious lung sounds.

- Crackles are produced upon immediate equalization of pressure in two compartments after airways have reopened. Crackles have been characterized as sounds like cellophane being crumpled

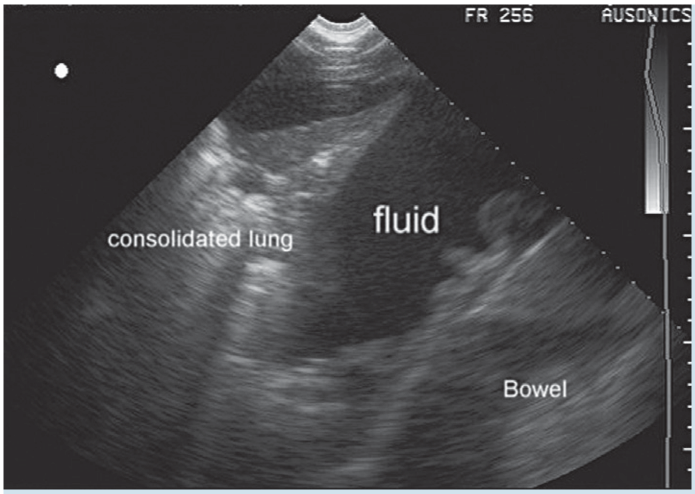


Figure 5.98: Complicated effusion causing respiratory distress in a 3-month-old colt. Note the excessive amount of pleural effusion.

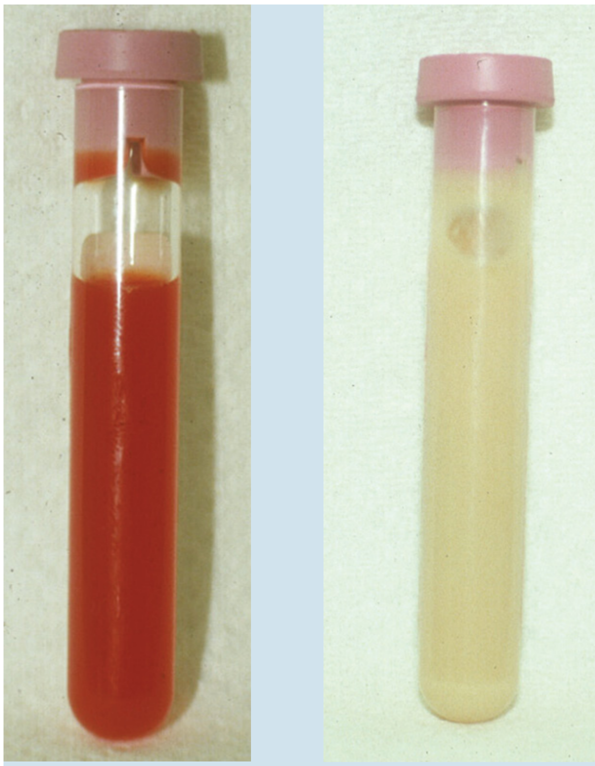


Figure 5.99: The tube on the left contains a serosanguinous pleural effusion obtained from the colt in Fig 5.98. The nucleated cell count was 22,000 with a glucose level of 25 mg/dL. These values are characteristic of a septic exudate. The tube on the right demonstrates an example of a purulent pleural exudate.

and are associated with obstructive disorders such as bronchopneumonia.
- Wheezes are associated with vibrations of airway walls and can be auscultated during inspiration or expiration.
- Wheezes are characterized as either monomorphic or polymorphic depending on the extent of the pneumonia.
- Pleural friction rubs are generated by the movement of visceral and parietal pleura which cross each other.

Figure 5.100: Indwelling chest tubes were placed in the foal in Fig 5.98 to permit continual drainage. Site selection is determined by ultrasonographic examination to allow placement in the location of the greatest accumulation of fluid.

- Percussion of the thorax consists of tapping the intercostal spaces of the thorax using a large spoon and a neurological hammer while evaluating the sound produced. Aerated tissues produce a resonant sound while consolidated lung, lung abscesses and bowel will produce a dull sound. Once the entire lung field has been percussed it is compared to that of a normal foal's lung field.
- Ultrasonography and radiography can help identify and characterize pathologic lesions. Culture and cytological evaluation via a tracheal wash can be performed to help determine the etiological bacteria involved.

Treatment

The selection of an antimicrobial regimen is based on:
- Identifying the causative agents:
  - common Gram-positive agents include *Streptococcus zooepidemicus*
  - common Gram-negative agents include *Actinobacillus equuli* and *Pasteurella* spp
  - determining the susceptibility patterns
  - host factors
  - tissue distribution of the drug.
- Foals with pneumonia secondary to severe septicemia require broad-spectrum therapy. Older foals with uncomplicated pneumonia can be initially treated with a Gram-positive regime with the understanding that if a poor response is noted then the institution of broad-spectrum therapy will be necessary.
- Anti-inflammatories are commonly used in foal pneumonia to help control fever and potentially alleviate inflammation of the lower respiratory tract.

Figure 5.101: Placement of an indwelling chest drain requires that the selected site be clipped and aseptically prepared. 10 mL of anesthetic is injected into the subcutaneous tissue and intercostal muscles. A stab incision is made through the skin large enough to accommodate the diameter of the large-bore chest tube selected. For most foals <6 months of age a 22–26 French chest tube will be adequate. This image is of an adult who has previously had an indwelling chest drain (note stitches at site of previous drain).

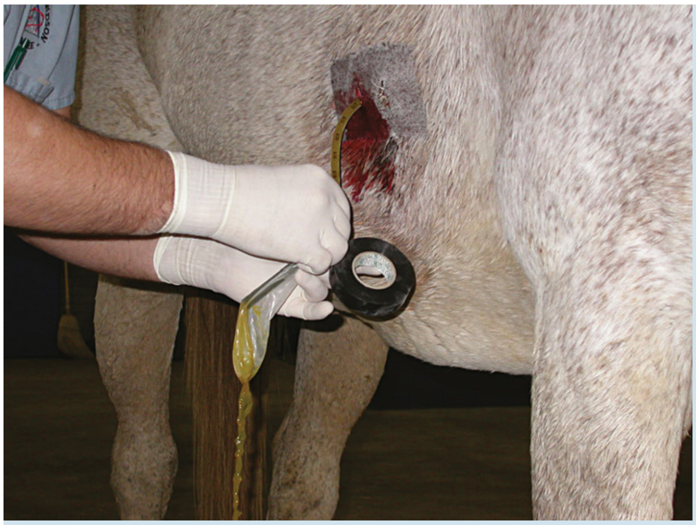


Figure 5.103: The trocar is removed from the tube and drainage of the effusion should occur immediately. Fluid can be collected for cytological evaluation and culture. A non-lubricated condom with the tip removed is placed at the end of the tube to allow unidirectional drainage. The drain should be monitored closely to ensure proper function so that aspiration of air and pneumothorax does not occur.

Figure 5.102: A moderate amount of force is required to push the tube through the skin, intercostal muscles, and parietal pleura into the pleural space. Note how the left hand is held firmly at the end of the trocar to prevent pushing the trocar into the underlying lung. A release of pressure is felt once the pleural cavity is entered.

Figure 5.104: A Chinese figure lock suture technique is used to secure the tube to the skin. The drain should be removed either when pleural exudate has stopped draining or when early indications of swelling and cellulitis have occurred.

- Foal pneumonia can also benefit from treatment with bronchodilators (see Hyperactive airway disease p. 165).
- It is very important to keep dust levels to a minimum in the foal's environment. This includes soaking all hay given to the mare and foal for 14 days and removing the foal from the stall when it is being cleaned.
- Pleural drainage **may** be warranted when the removal of exudate and debris from the pleural space allows for the re-expansion of the lung. Decisions regarding pleural drainage are based on classification of the pleural fluid as a complicated or uncomplicated effusion:
  - Uncomplicated effusions are those that do not have sufficient volume to cause respiratory distress and are classified as a transudate which contains less than 10,000 nucleated cells per μL and less than 2.5 g/dL of protein. Non-degenerative neutrophils are the primary cells seen, mononuclear cells (macrophages, lymphocytes) are the second most common.
  - Complicated effusions are characterized as having a sufficient volume to cause respiratory distress, greater than 10,000 nucleated per μl with increased numbers of degenerative neutrophils, greater than 2.5 g/dL of protein, pH < 7.2, glucose concentration <40 mg/dL, putrid odor, cytologically visible bacteria or positive culture results. Most complicated effusions require pleural drainage.

Figure 5.105: One time thoracocentesis of one or both sides of the thoracic cavity may be achieved by the use of a teat cannula. This has the advantage of requiring a smaller incision and is particularly useful in neonates. Here the teat cannula has been attached to a syringe via an extension set to prevent air entering the thoracic cavity. Ideally a three-way stopcock should also be in place.

Figure 5.106: The use of the Equine Haler^{TM}. This device distributes 9% of the inhaled drug into the lower airways compared to 15% in humans (courtesy of Jorvet Laboratories, Loveland, Colorado, USA).

Juvenile hyperactive airway disease (Figs 5.106–5.108)

This disease tends to affect foals 4–7 months of age and is poorly understood. It appears that juvenile hyper-reactive airway disease is a syndrome of small airway disease that has minimal response to antibiotics alone. The majority of the cases in the United States present between June and September.

Clinical signs

Clinical signs are consistent:

- Foals present with a history of tachypnea with an exaggerated abdominal component.
- A tracheal rattle and flared nostrils with minimal air flow is a common clinical finding. Auscultation of the chest reveals polyphonic crackles and wheezes associated with the marked bronchoconstriction and excessive mucus production that accompanies this disease.

Diagnosis

Clinical signs are in excess of the diagnostic results:

- Minimal growth is noted on the transtracheal wash.
- The complete white blood cell count is typically normal with a normal fibrinogen level. Signs tend to persist despite broad-spectrum antimicrobial treatment.
- Thoracic ultrasonography is usually unrewarding revealing minimal pleural irregularities (comet tails).
- Thoracic radiographs reveal thickened bronchioles and a mild interstitial pattern.

Treatment

Treatment is aimed at resolving cyanosis with intranasal oxygen, bronchodilators and reducing the inflammation within the lung. Foals should be placed on 7–10 L/min of oxygen via a nasal cannula.

Bronchodilators are used to relieve the obstruction of the small airways caused by airway smooth muscle contraction. The administration of bronchodilators (Figs 5.106 & 5.107) should always be combined with strict environmental dust control (soaking hay and removing from stall/barn when cleaning) and corticosteroid therapy to help reduce the inflammation of the lower airways.

- Various orally or parenterally administered bronchodilators include:
  - Clenbuterol (0.8–3.2 μg/kg PO BID) is an excellent bronchodilator with mucokinetic properties to help clear mucus from the airways.
  - Aminophylline (5–10 mg/kg diluted in fluids can be given BID to QID IV) is a xanthine derivative that is a bronchodilator, enhances mucociliary clearance, contractility of the diaphragm and delays fatigue of the muscles responsible for respiration.
  - Glycopyrrolate is used as a "rescue" drug when immediate bronchodilation is needed. It is a synthetic antimuscarinic agent that blocks the M_3-muscarinic receptors and causes bronchodilation (0.002–0.007 mg/kg IV once).
- Inhalation Bronchodilators *(during severe airway obstruction these drugs have poor pulmonary distribution)*. Depending on the formulation used, inhaled bronchodilators may be administered by nebulization or by inhaler.
  - Albuterol is a beta$_2$ agonist which serves as a rescue therapy. This medication can improve pulmonary function by 70% within 5 minutes of administration. Unfortunately the effect only lasts 1–3 hours:
    - give 3–6 activations (450 μg) 2× daily
    - nebulize 2–5 mg (as a rescue dose for severe bronchoconstriction the author has performed nebulization every 15 min for 1 hour and then every 4–6 hours until bronchoconstriction has resolved)
  - Ipratropium bromide:
    - 3 activations 2× daily
    - nebulize 1 vial (500 μg) 3–4× daily
- Systemic dexamethasone has been demonstrated to improve lung function within hours of administration with a maximal response

Figure 5.107: Nebulization of a foal suffering from juvenile hyper-reactive respiratory disease. The foal is being nebulized with albuterol and ipratropium. A nebulizing mask can be easily made with a milk jug and proper padding to help decrease loss of the medication into the surrounding environment.

Figure 5.108: A typical "acorn" nebulizer used to administer albuterol and ipratropium to the foal in Fig 5.107.

obtained by day 7. Steroids not only reduce inflammation within the chest but over time can up-regulate the number of $beta_2$ receptors within the airways. Dexamethasone is administered intravenously at a rate of 0.1 mg/kg q24hr and then the dose is decreased to 0.05 mg/kg q24hr for another 4–5 days after clinical signs resolve. Inhaled steroid therapy can also be used in conjunction with systemic steroids.

- Inhaled steroids may be administered for several weeks after the systemic steroid therapy has been discontinued. Examples of inhaled steroids in foals include:
  - Fluticasone (220 μg/activation): 4–8 activations once a day for 2 weeks and then every other day for another 2 weeks
  - Beclomethasone dipropionate (80 μg/activation): 5–8 activations once a day for 2 weeks and then every other day for another 2 weeks.

Recommended reading

Pusterla N et al 2006 Infectious respiratory diseases. Clinical Techniques in Equine Practice 5(3): 174–186

Schambourg MA et al 2003 Thoracic trauma in foals: post mortem findings. Equine Veterinary Journal 5:78–81

Slovis NM 2004 Atlas of equine endoscopy. Mosby, St Louis

Wilson WD 2003 Foal pneumonia. In: Robinson NE (ed) Current therapy in equine medicine, 5th edn. Saunders, Philadelphia, p 666–673

Reed SM, Bayly WM, Sellon DC 2004 Equine internal medicine. Saunders, Philadelphia

CHAPTER 6

The urinary system

Kim A. Sprayberry DVM, DACVIM

Introduction

When clinical signs of disease referable to the urogenital tract are observed in the perinatal foal, an extensive physical exam should be performed. In most instances, though, the physical examination will comprise only part of a more comprehensive diagnostic work-up, because many of the problems that cause urogenital tract dysfunction in foals are internal and a physical exam alone is unlikely to allow the practitioner to arrive at a definitive diagnosis.

Referral to an equine inpatient clinic is not always necessary to perform a complete diagnostic work-up for a foal with signs of oliguria, anuria, stranguria, or azotemia, given the increasing use of diagnostic ultrasound and point-of-care analytical equipment by equine practitioners. But whether the necessary testing and intervention are undertaken in the field or in a hospital setting, a comprehensive diagnostic plan for such foals should include:

- careful consideration of the medical and birth history
- clinical assessment
- CBC and serum biochemical profile that includes determination of BUN, creatinine, and IgG (if the foal is a neonate)
- urinalysis
- ultrasonographic imaging of the foal's thorax and abdomen, with particular scrutiny directed to the urinary tract and umbilical remnants
- radiography, if ultrasound is not available
- possibly paracentesis.

Whether foals affected with urinary tract dysfunction have congenital or acquired disease, such a work-up is likely to yield information that will lead to a definitive diagnosis. As is true for most disease conditions involving perinatal foals, promptness in evaluation and initiation of treatment are key to successful outcomes.

History and physical examination findings (Figs 6.1 & 6.2)

- Deriving usable information from an owner's recounting of a foal's medical history can justifiably be challenging; it can be difficult even for veterinarians to distinguish between a foal that is straining to urinate and a foal that is straining to defecate. Foals straining to urinate typically assume a body position in which the back is ventroflexed and the hind limbs are placed caudal to the pelvis (i.e. placed farther away from the forelimbs); this posture is commonly observed in foals with uroperitoneum.
- If the foaling was unattended or no postpartum mare–foal examination (including inspection of the placental membranes) was performed, it may be impossible to obtain certain pieces of information that would otherwise be valuable in formulating a list of likely differential diagnoses.
- With neonatal foals, signs of urinary tract dysfunction should be interpreted in light of the broader possibility that sepsis is the underlying problem, and that diagnosis must be evaluated and ruled in or out. This is important not only from a diagnostic perspective but also for relating to the owner information pertaining to estimated costs and prognosis.
- When querying owners or farm managers about a foal's medical history, prior treatment of the foal with common nephrotoxic drugs such as the aminoglycoside antimicrobials, oxytetracycline, polymyxin, NSAIDs, and parenterally-administered vitamin D or K should be determined.
- The foal's hydration status at the time of examination and whether earlier episodes of dehydration were observed are important. The latter should be suspected if owners report that the foal did not rise and nurse within 3 hours of delivery, the mare did not permit the

foal to nurse, or if "milk face" (i.e. milk stuck to the hair on the poll or forehead, signaling that the foal assumed the nursing position but was unwilling or unable to suckle) was observed (see Fig 4.74).
- Historical or ongoing urine discoloration may indicate hematuria, hemoglobinuria, or myoglobinuria secondary to various conditions, and subsequent pigment-associated nephropathy.

Laboratory findings

Azotemia (Figs 6.3 & 6.4)

- Azotemia refers to an increase in blood urea nitrogen (BUN) and creatinine in plasma or serum.
- Clinical signs typically observed in foals with azotemia include lethargy, inappetance, and dehydration (manifested as some combination of enophthalmos, decreased skin turgor, poor jugular distensibility, weak pulse quality, and tachycardia).
- Azotemic foals, like adult horses, may have pre-renal, intrinsic renal, or post-renal conditions. Pre-renal conditions, such as dehydration or circulating volume depletion, intrinsic renal disease such as congenital malformations or septic infarcts, and post-renal conditions such as lower-tract obstruction or bladder rupture with consequent uroperitoneum, all result in azotemia. Thus increased values for BUN and creatinine are non-specific indicators that function of the urinary tract is compromised.
- Such signs are non-specific in character; accompanying changes in blood work and imaging studies are often needed to complete the diagnostic plan.
- ***High serum BUN or creatinine concentrations (in the range of 3.0–25.0 mg/dL) in foals that are approximately 48 hours of age or younger are not uncommon, and may reflect placental compromise or failure***; this form of *non-renal azotemia* in the foal should prompt examination of the placental membranes, if available, because placentitis is a possible finding. Even if the placenta is not available for examination, dams of azotemic newborn foals should

Figure 6.1: Filly urinating normally. Note there is no ventroflexion of the back.

Figure 6.2: Stranguria. Note ventroflexion of back and caudal position of hindlegs compared to (Fig 6.1).

Figure 6.3: Enophthalmus (sunken eye) in a severely dehydrated foal. This foal was suffering from dehydration secondary to diarrhea. This is an example of a pre-renal cause of azotemia.

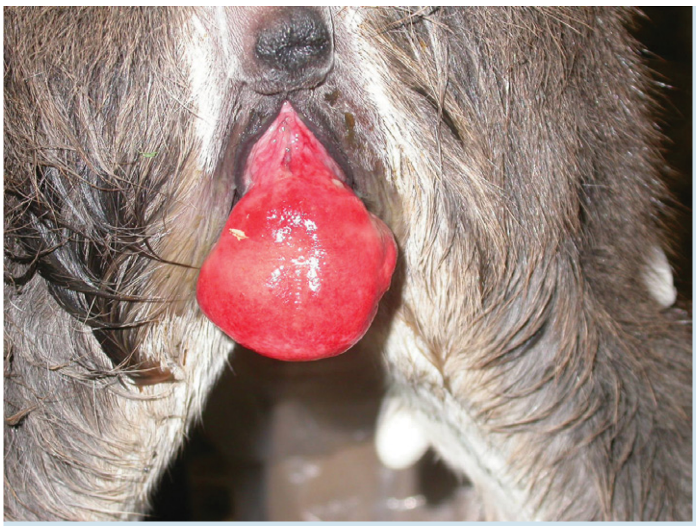


Figure 6.4: Urinary obstruction secondary to bladder prolapse and eversion. This foal was suffering from cystitis which resulted in stranguria. This is an example of a post-renal cause of azotemia.

have a systemic and reproductive tract examination and a CBC and fibrinogen determination. In the author's experience, detection of sepsis or non-renal azotemia in the neonate is usually accompanied by concurrent abnormalities, clinical or subclinical, in the dam, and physical examination, CBC and serum biochemical profile, and uterine lavage are recommended for dams of ill foals or foals that appear clinically healthy but are azotemic.

- Foals with postpartum azotemia should be monitored very closely for clinical or hematologic signs of sepsis during the first week after birth because placentitis, whether or not it was recognized prepartum, is a possible cause of placental dysfunction and non-renal azotemia in newborn foals.

Alterations in electrolytes

- The tubular segments of the nephron are responsible for conservation and excretion of sodium and potassium, respectively.
- Hence, with clinically significant oliguria or nephrosis, refractory hyponatremia or life-threatening hyperkalemia may develop.
- The metabolic derangements that occur can result in clinical signs other than those associated with azotemia. Hyponatremia can result in neurological disturbances including convulsions; progressive hyperkalemia results in cardiac dysrhythmias. Lethargy, depression and a loss of interest in nursing develop as the metabolic status deteriorates.
- Severe hyponatremia in the absence of diarrheic enterocolitis and attendant fluid and electrolyte losses should prompt suspicion of tubular damage and impairment of sodium reabsorption.
- Severe hyperkalemia should be managed with administration of dextrose- or glucose-containing fluids, sodium bicarbonate solution (administration of any bicarbonate-containing solution should only be initiated in foals without impaired pulmonary gas exchange) and calcium gluconate solution.
- Uroperitoneum secondary to rupture or functional leaking of the ureters, bladder, or urachus results in classic changes of hyponatremia, hypochloremia, hyperkalemia, and azotemia.

Hemogram

- Abnormalities in the leukocyte count, whether high or low, are highly informative; findings of neutropenia, with or without failure of passive transfer (i.e. low serum IgG concentration), are grounds for diagnosing sepsis as an underlying or complicating condition, whereas a high neutrophil count, elevated fibrinogen concentration, or both suggest that there is a pyogenic inflammatory focus of some chronicity. Manifestations of the latter type of condition most commonly involve infections in the foal's thorax, abdomen, or joints.
- Septic showering can occur in association with bacterial sepsis of multiple etiologies (including Gram-positive organisms) but bacteremia with the Gram-negative pathogen *Actinobacillus equuli* has been particularly associated with infection of the kidneys and liver in neonatal foals.
- Aseptic collection of blood for microbial culture is recommended in foals with neutropenia and low serum IgG concentrations that have concurrent signs of urinary tract dysfunction (see Fig 3.68).

Urinalysis

- Because of their all-liquid diet, the urine of neonatal foals is more dilute than that of adult horses, with normal specific gravity in the range of 1.001–1.015 and a reference range for urine osmolarity of 102 ± 24 mOsm/L.
- In contrast to the alkaline pH of urine in healthy adult horses, neonatal foal urine is typically more acidic, with pH in the range of 6.0–7.0, also a reflection of the milk (versus forage) diet.
- Healthy foals nurse frequently and consequently, urinate frequently; *daily urine output in* ***healthy*** *foals is approximately 130–150 mL/kg.*
- Values for specific gravity and volumes of urine produced in ill foals, particularly those receiving intravenous fluid support, will vary.
- Urinalysis is also used for determining fractional excretion ratios of electrolytes and enzymuria. Alterations in the fractional excretion of sodium and increases in the urinary GGT-to-serum creatinine concentration ratio are earlier indicators of tubular damage than is serum creatinine alone. Fractional excretion of sodium should be <1% in healthy sucking foals and is <0.5% in most foals. The GGT : serum creatinine ratio in healthy perinatal foals should be ≤46.5.

Diagnostic imaging

Ultrasound (Figs 6.5–6.9)

Use of diagnostic ultrasound vastly enhances the ability of an examiner to render medical and surgical diagnoses in foals, and its importance as a component of the comprehensive examination of foals cannot be overstated.

Table 6.1: Normal ranges for the urinalysis of the equine neonate

Specific Gravity	1.001–1.015
pH	6.0–7.0
Protein	Negative to +30
Glucose	Negative
Crystals	None (rarely calcium oxalate)
Casts	Negative
Nitrate	Negative
Red blood cells	Negative
White blood cells	+3/HPF
Bacteria	None in a catheterized sample

Normal foal values are distinctly different than adults.
Foals up to 36 hours have proteinuria because of colostral aborption and subsequent protein excretion.

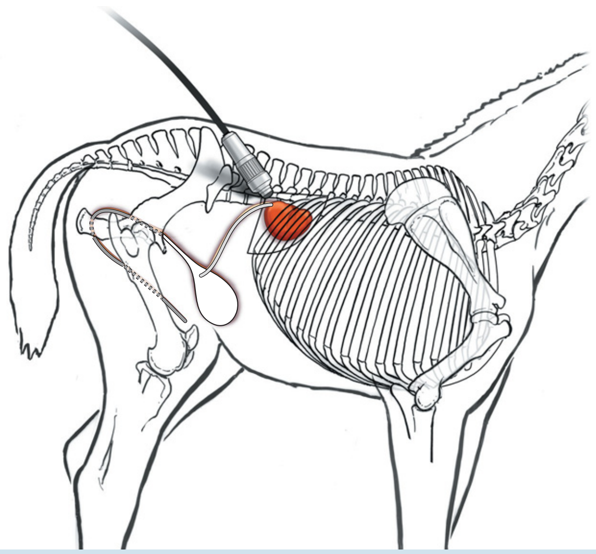


Figure 6.5: Schematic image of ultrasonography of the right kidney (line drawing from Divers TJ, Perkins G 2003 Urinary and hepatic disorders in neonatal foals. Clinical Techniques in Equine Practice 2:67–78).

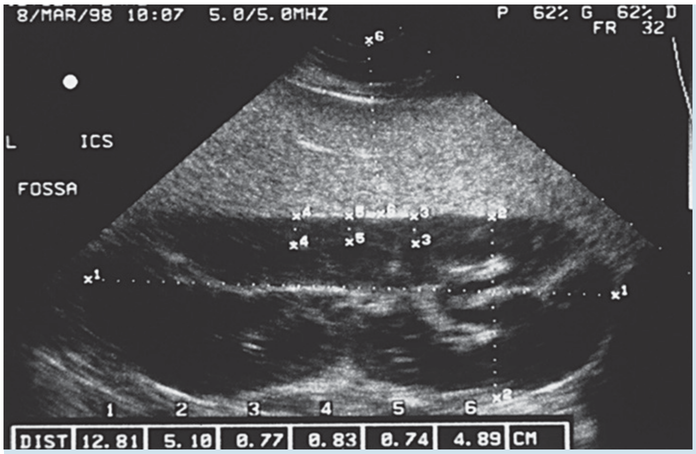


Figure 6.6: Normal ultrasound of left kidney (longitudinal view) in a 1-month-old QH with measurements for length (1) and width (2) of the kidney, measurements of cortical thickness (3, 4 & 5) and distance from the body surface (6). Note the spleen at the top of the image.

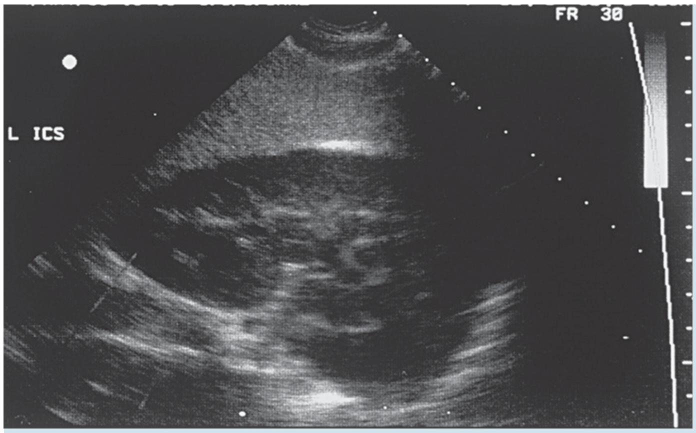


Figure 6.7: Normal left kidney in a 7-day-old Standardbred filly, cross-sectional view.

Because of the small size of neonatal equine patients, imaging studies are usually highly productive and yield the visual information that, together with the information contained in the CBC and serum biochemical analyses, can lead the practitioner to a definitive diagnosis.

Imaging is particularly important in foals which are already being treated for other problems, especially in the instance of hospitalized foals. In such foals, the metabolic derangements associated with conditions such as uroperitoneum (i.e. hyponatremia, hypochloremia, hyperkalemia, metabolic acidosis, and azotemia) may be mild or even non-existent as a result of the foal receiving intravenous fluid support or other treatments which would tend to palliate the severity of changes that might otherwise develop. Foals which are recumbent secondary to neurologic complications of hypoxic–ischemic encephalopathy or sepsis may be at increased risk of developing ruptured bladder, irrespective of age or sex. Likewise, the forces of abdominal press (tenesmus) in a foal that is persistently straining because of meconium impaction or fecal obstipation may lead to re-opening of a previously closed urachus. Therefore, the combination of results of blood work and diagnostic imaging are extremely helpful to the equine practitioner, both in field and clinical settings.

The right kidney is located ventral to the transverse spinous processes between the 14^{th} and 16^{th} intercostal spaces (ICS) from 2 cm

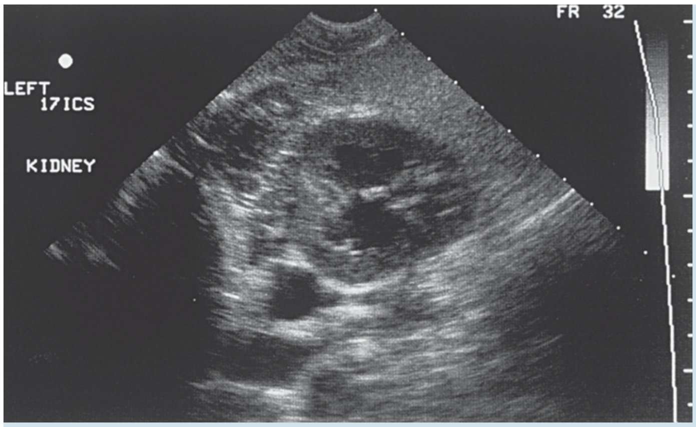


Figure 6.8: Normal left kidney, transverse view with only one lobe visible in a 14-day-old Standardbred filly. Note the spleen at the top of the image.

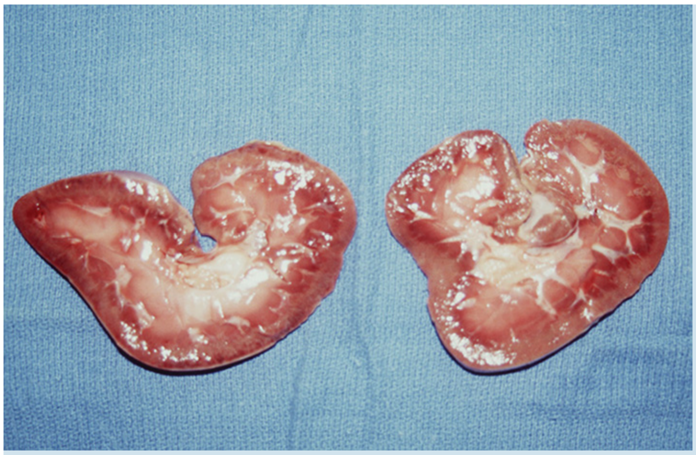


Figure 6.9: Cross-section of a normal kidney at postmortem. Right is on the right and left is on the left.

dorsal to the tuber coxae to 12 cm ventral. The right kidney has a heart-shaped appearance. The left kidney has a bean-shaped appearance and is located from the 15^{th} ICS to the caudal border of the left paralumbar fossa and from 2 cm dorsal to 15 cm ventral to the dorsal margin of the tuber coxae. Measurement of kidney length and width in foals up to 6 months of age (Table 6.2) showed a significant difference between right and left kidneys, with the left kidney being longer and the right kidney being wider for all age groups. The renal pelvis and calices are hyperechoic, the renal cortex is hypoechoic in relation to the renal calices and the renal medullary pyramids are relatively anechoic, allowing for a prominent corticomedullary junction. The renal capsule is imaged as a thin hyperechoic line surrounding the kidney.

Congenital or inherited disorders

History

Congenital renal diseases such as renal agenesis, renal hypoplasia, and renal dysplasia cause primary or intrinsic renal disease.

Table 6.2. Kidney length and width in foals

Age	<1 month	1–2 months	2–3 months	3–4 months	4–5 months	5–6 months
Right kidney length	7.5–10	8.2–9.9	8.8–10.7	9.5–10.8	10.3–11.3	10.7–11.8
Right kidney width	9.0–12.4	11.3–13.1	12.0–14.6	12.1–14.7	12.8–15.4	13.5–15.6
Left kidney length	9.8–13.4	11.8–15.0	12.5–15.6	13.3–15.7	14.0–16.8	14.8–16.9
Left kidney width	6.9–9.3	8.6–9.5	8.7–10.2	9.3–10.6	9.8–11.0	10.2–11.0

Clinical signs

- Foals with congenital renal malformation or agenesis may or may not have signs of renal failure dependant on whether or not both kidneys are affected.
- Clinical findings that point to renal failure are persistent azotemia in a euvolemic state, electrolyte derangements such as hyponatremia or hyperkalemia, and increased fractional excretion of sodium (i.e. values >1%). However, the biochemical findings may be subtle depending on the degree of deformity. *Frequently the first clinical sign that is noted is that the foal is growing poorly.*
- In foals with renal failure as a result of intrinsic renal disease, serum creatinine concentration will not decrease in the face of intravenous fluid and diuresis.
- Imaging studies, especially ultrasonographic, can be highly informative in foals with developmental anomalies of the urinary tract. Observations of abnormal renal appearance or missing kidneys are suggestive of congenital disease, and the more invasive diagnostic tests (biopsy) can be recommended if a conclusive diagnosis is desired.

Diagnosis

- Foals affected with the following congenital renal diseases typically have a similar clinical appearance, and may survive to adulthood before the conditions are detected.
- Differentiation among these diseases is made on the basis of histologic evaluation.

Renal agenesis

- The congenital absence of major organs is rare in all species.
- In foals with suspected renal agenesis, careful necropsy examination, including histological evaluation of tissues from surrounding anatomic regions, may reveal rudiments of the missing tissue.
- Whether the condition is genetic or arises secondary to developmental accident during organogenesis is unclear.
- In a report involving a 4-year-old Quarter Horse gelding, unilateral renal agenesis was accompanied by urogenital abnormalities on the contralateral side.
- Compensatory hypertrophy of the existing kidney in the congenital absence of one kidney has been reported in 12 horses.
- Azotemia in an affected foal means that function of the single kidney is impaired, and the prognosis for long-term survival is poor.

Renal dysplasia

- Renal dysplasia is the result of disruption of the processes of renal organogenesis.

Figure 6.10: A 3-month-old foal diagnosed with renal hypoplasia and suspected renal dysplasia ante-mortem on the basis of ultrasonographic findings. A renal condition was initially suspected on the basis of poor growth and a mild consistent azotemia despite fluid therapy. Postmortem examination confirmed a hypoplastic left kidney and dysplasia of both kidneys.

- The abnormal renal tissue is composed of immature and disorganized tissue, and diagnosis is made on the basis of histological evaluation of a renal biopsy specimen, although some dysplastic kidneys may be grossly abnormal.
- Sonographic imaging reveals dysplastic kidneys to be small and misshapen. The renal cortices are hyperechoic and thickened. Corticomedullary junctions are indistinct, and the corticomedullary ratio is high.
- Increased echogenicity in kidneys is usually correlated with fibrotic change and such observations in foals with clinical signs of intrinsic renal disease are grounds for recommending a renal biopsy procedure.

Renal hypoplasia (Figs 6.10–6.14)

- Renal hypoplasia is diagnosed when one kidney is at least 50% smaller than normal or when the total renal mass is decreased by more than one-third. Gross and histological examination of affected kidneys reveals decreased cortical and medullary tissue.
- Kidneys may be so small that they are difficult or impossible to image via ultrasonography or contrast cystography, and renal agenesis may mistakenly be diagnosed.
- Diagnosis is made via histologic evaluation of a renal biopsy specimen.
- Dysplastic changes in the hypoplastic kidney are also common.

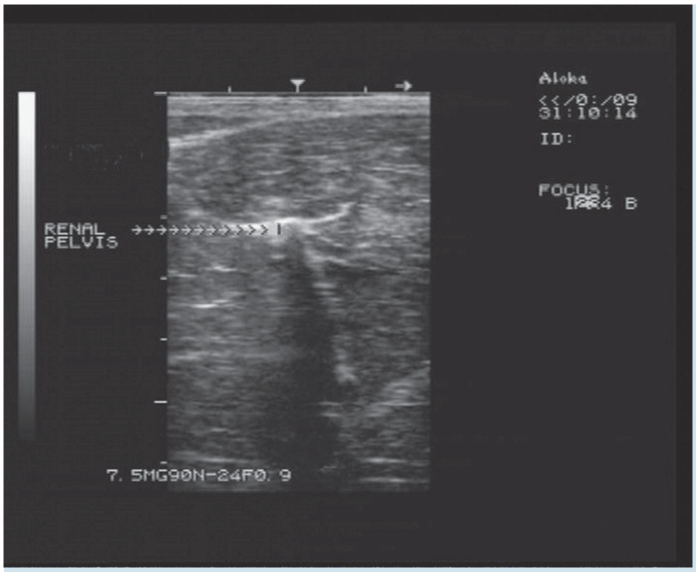


Figure 6.11: Ultrasonographic image of the hypoplastic left kidney (same foal as Fig 6.10). It is difficult to discern any normal tissue with no evidence of a corticomedullary junction. The hyperechoic tissue in the center was judged to be the renal pelvis.

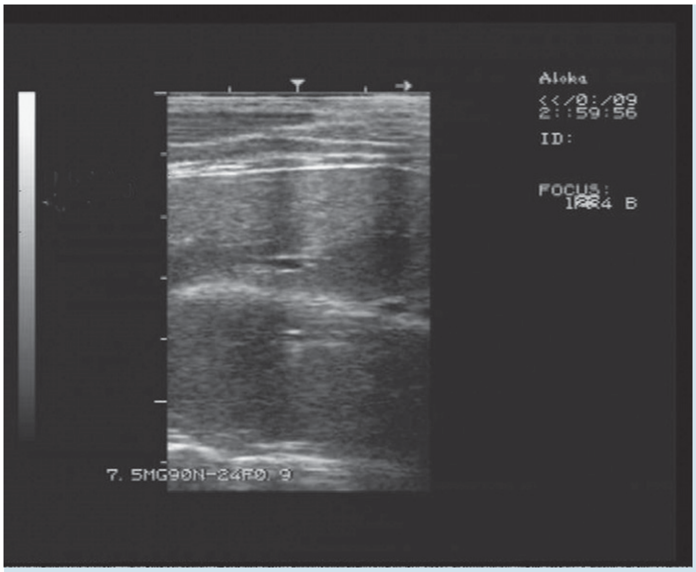
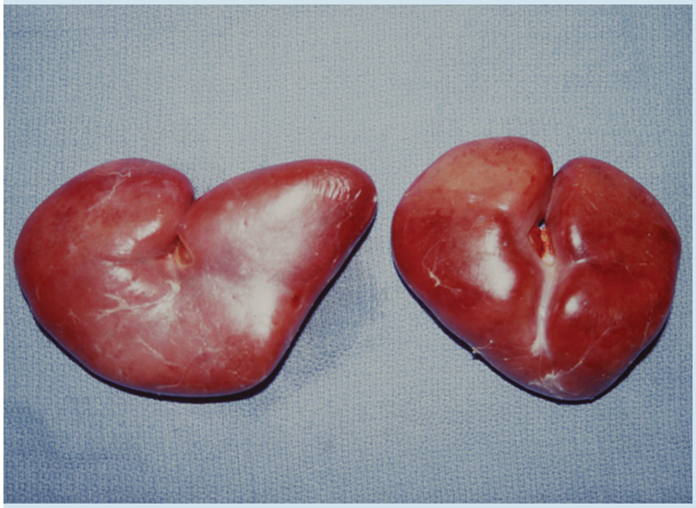


Figure 6.12: Ultrasonographic image of the right kidney from foal in Fig 6.10. Although this organ is more clearly a kidney with an apparent corticomedullary junction there is an overall increase in echogenicity and abnormal shape.

Renal cysts and polycystic renal disease (Figs 6.15 & 6.16)

- These conditions may also be diagnosed with reasonable certainty on the basis of ultrasonographic imaging findings.
- Foals with single small cysts (<3 cm) may be healthy with no other associated clinical signs. Single cysts may be discovered incidentally during ultrasound examination undertaken for other reasons. Foals with multiple small cysts or single large cysts, like foals with other congenital renal malformations, will have clinical signs of renal



Figures 6.13 & 6.14: Left and right kidneys from the foal in Figs 6.10–12. Note the very small and misshapen left kidney and the misshapen right kidney when compared to normal foal kidneys in Fig 6.14. In both images the right kidney is on the right side of the image and the left kidney is on the left side of the image.

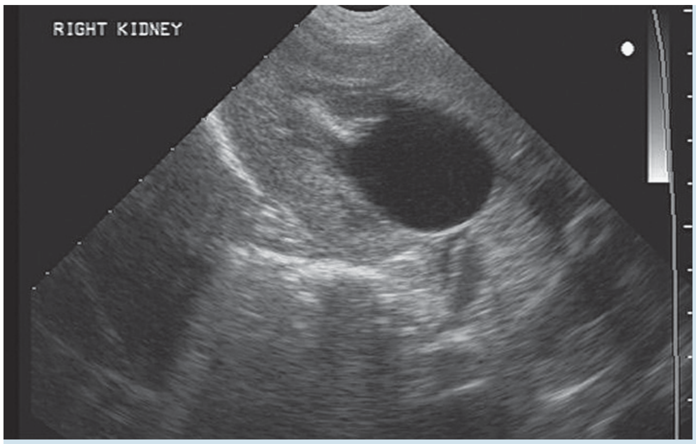


Figure 6.15: Ultrasonographic image of a single renal cyst found incidentally during examination of a foal with colitis. This should not be confused with hydronephrosis which is a fluid dilation of the renal pelvis (see Fig 6.17).

Figure 6.16: Single large renal cyst found incidentally during a postmortem examination.

failure. Results of laboratory work and failure to respond favorably to treatment are indications of intrinsic renal disease.

- Upon imaging, overall dimensions of the kidneys may be enlarged, and the cortex and medulla are replaced by one or multiple thin-walled cysts that are typically approximately 1.5 cm in diameter and filled with anechoic or slightly echogenic fluid.
- Although the pathogenesis of polycystic kidney disease in horses is unknown, the condition is heritable as an autosomal dominant or recessive trait in humans.
- Histologic analysis of polycystic kidneys in humans reveals cysts to represent dilatations of the proximal or distal tubules. Other histologic findings in affected equine kidneys include severe alterations in cortical architecture, nephron sclerosis, tubular dilatation, and cytomegalic tubular epithelial cells.
- Extensive diagnostic efforts are typically not pursued in foals in which polycystic kidneys are detected because the prognosis for survival is poor.
- Including ultrasonographic imaging as part of routine postpartum foal examinations permits practitioners to detect such abnormalities as early as possible and is strongly recommended for foals which the owner intends to insure or when the breeding contract specifies that contractual obligations have been met upon delivery of a healthy foal.

Hydronephrosis (Figs 6.17–6.21)

- Detection of hydronephrosis in foals usually occurs during ultrasonographic imaging of the abdomen.
- Hydronephrosis develops secondary to mechanical obstruction or functional obstruction (failure of ureteral peristalsis) of some portion of the urinary tract, and as such, should be thought of as the result of a problem rather than a primary diagnosis.
- In adult horses, the most common cause of hydronephrosis is urolithiasis, in the form of cystic calculi, nephrolithiasis, or ureterolithiasis. But in foals, dilatation of the renal pelvis or ureter usually develops secondary to obstruction of some segment of the lower portion of the urinary tract by blood clots, by ectopic ureters, or, uncommonly, by vascular anomalies involving a connection

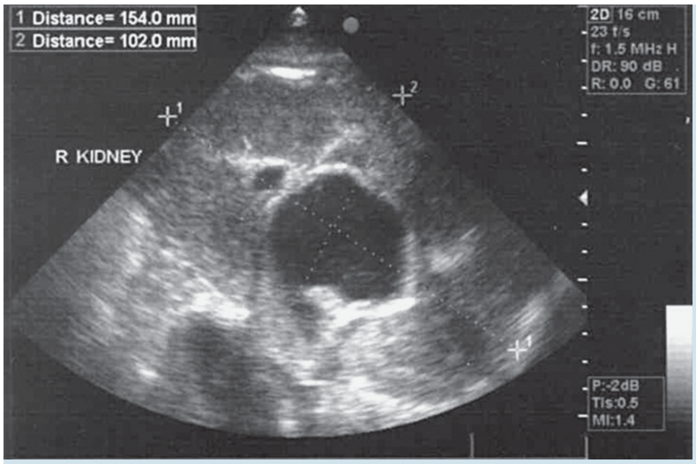


Figure 6.17: Ultrasound image of a right kidney with hydronephrosis; note the enlarged, fluid-filled renal pelvis.

Figure 6.18: Bilateral hydronephrosis; note the enlarged ureters.

between vascular structures and a ureter. One of the editors (NMS) has seen six foals presented with hydronephrosis/hydroureter secondary to posterior urethral valves (PUV).

- Mucus plugs in the urethra of male foals have been reported and result in bladder distension and concomitant bilateral ureteral distension. That condition, however, is acute in nature and results in clinical signs of colic and stranguria.
- Hydronephrosis itself does not typically elicit specific clinical signs.
- Particularly in hospitalized foals receiving intravenous fluid support or treatment with diuretics, *it is important to differentiate hydronephrosis from pyelectasia, the condition of enlarged renal pelvis and ureters arising secondary to diuresis of large volumes of intravenous fluids.* Pyelectasia is diagnosed by means of sonographic detection of dilatated urinary structures but no obstructing lesions. The condition should be reversible with appropriate modulation of the intravenous fluid administration.
- In foals with functional ureteral obstruction, catheterization of the ureters and/or balloon dilation resulted in resolution of the dilatation and recovery of normal function.
- Foals with hydronephrosis may or may not be azotemic.

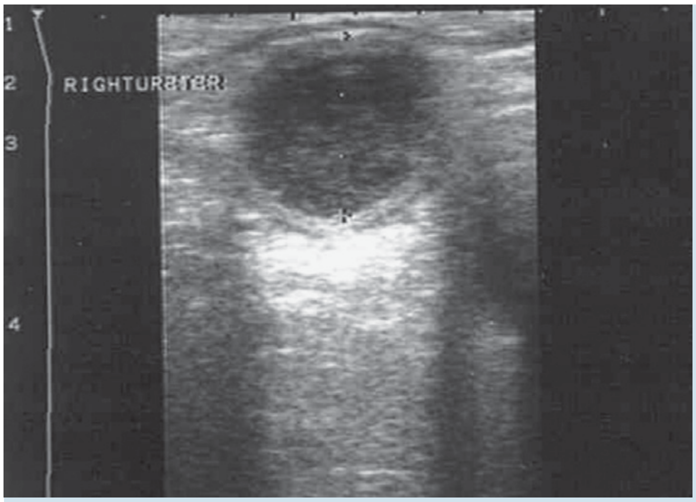
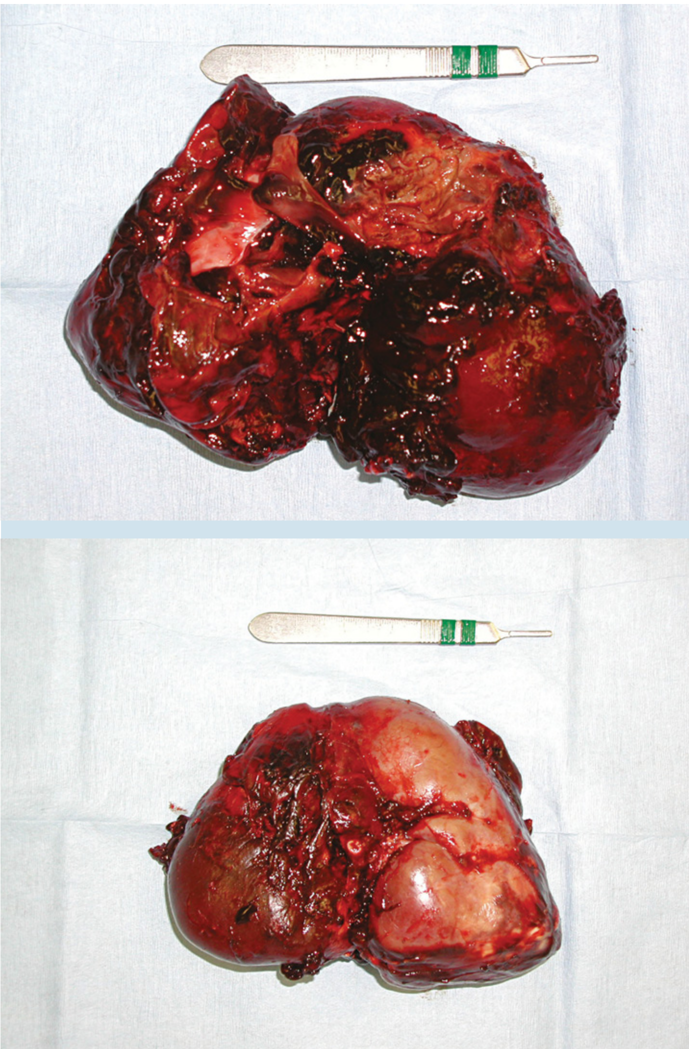


Figure 6.19: Ultrasound image of hydroureter.



Figures 6.20 & 6.21: Marked renal hemorrhage with clot formation as a result of traumatic injury. Such clot formation would likely result in urinary tract obstruction.

- If urinary tract obstruction is arising as a result of a blood clot, clinical or occult hematuria may be revealed on urinalysis.
- Whereas diagnostic ultrasonography is the definitive means of diagnosis of hydronephrosis, identifying the cause of obstruction may necessitate endoscopic evaluation of the urethra and bladder. Vaginal speculum examination may be used to diagnose ectopic ureters in fillies.
- Blood clots may form in the kidneys or ureters following abdominal injury, such as might occur if the mare steps on or kicks the foal. Stabling mare–foal pairs in larger stalls may help minimize the likelihood of accidental injuries.

Differential diagnoses

When dilatation of the renal pelvis or ureter is visualized via ultrasonography or endoscopy, the most common conditions that should be ruled out include:

- renal cyst or multicystic kidneys
- dysplastic kidneys
- renal agenesis (the existing kidney may appear hypertrophic)
- pyelectasia.

It is important to remember that dilatation of one or both renal pelves and ureters should prompt careful assessment of the rest of the urinary tract to detect an obstruction.

Diagnostic tests

- Diagnostic ultrasound
- Endoscopic examination
- Serum biochemistry: possible azotemia
- Urinalysis: possible hematuria (with or without anemia)

Treatment

- Treatment for hydronephrosis depends on the cause of obstruction. Supportive diuresis and serial sonographic monitoring may be all that is necessary in some instances.
- Surgical correction of ectopic ureters may be considered if the foal's value warrants such intervention.
- Broad-spectrum antimicrobials should be administered if hematomas are detected as the cause of urinary tract obstruction.
- Pyelectasia typically resolves promptly when the rate and volume of fluids administered are decreased.

Ectopic ureters

- The ureters course from the renal hilus and penetrate the dorsal wall of the bladder at the trigone at an oblique angle of entry. The trigone is the area just craniad to the neck of the bladder and proximal portion of the urethra in anatomically normal horses and foals.
- The so-called internal urethral sphincter refers to the segment in the proximal portion of the urethra where the smooth muscle layer is augmented with fascicles of skeletal muscle, forming a functional sphincter (which is histologically, but not grossly, visible) that is under voluntary control.
- In individuals with ectopic ureters, one or both ureteral ostia empties into the bladder or urethra at some point distal to the functional sphincter. In some animals, the aberrant ureters may open into the uterine lumen or vaginal tract.

Diagnosis and treatment

- The diagnosis may be suspected on the basis of clinical signs such as urine dribbling, but definitive diagnosis requires endoscopy or cystography.
- This malformation can only be corrected by means of reconstructive surgery or, if the condition is unilateral, nephrectomy. Reconstructive surgery is rarely successful as it is technically difficult to perform and post-surgical complications such as renal infections are common. Nephrectomy is also a difficult surgical procedure but the success rate is higher with this approach.

Patent urachus (Figs 6.22–6.27)

- The urachus is the conduit through which fluid waste is conducted from the fetal urinary bladder to the allantoic space, and extends from the bladder apex to the umbilicus.
- Normal closure of the structure at parturition leads to distension of the bladder and initiation of the neurologic reflexes that prompt the foal to assume micturition posture and urinate. Failure of the urachus to close leads to dribbling or streaming of urine from the umbilical stump, even though affected foals typically posture and urinate through the urethra also.
- The paired umbilical arteries become the left and right round ligaments of the bladder; these structures are enclosed in folds of peritoneum called the lateral ligaments of the bladder.
- The umbilical vein becomes the round ligament of the liver – a cord-like structure surrounded by the falciform ligament.
- The urachus is not a discrete, tubular structure, but rather is the potential space between the two umbilical arteries and that is circumscribed by a thin membrane that surrounds that segment of the arteries coursing from the bladder apex to the umbilicus.
- Foals may be born with the urachus normally closed but then be observed dribbling urine from the external umbilical remnant on the first or second day of postpartum life. This may occur in foals that are constipated or have meconium impaction and strain to defecate; the intra-abdominal pressure created by abdominal press can lead to re-opening of the urachal lumen and the exit of urine via the urachal remnant. Re-opening of the urachus may also occur as a result of infection and is also frequently seen in foals that are largely recumbent for other reasons, e.g. septicemia.

Figure 6.22: Patent urachus in a 13-day-old foal suffering from colitis.

Diagnosis

The observation of urine dribbling from the umbilical stump of a young foal is the basis for making a diagnosis of patent urachus, although sonographic imaging may be used to confirm the patent status of the structure internally and is a good tool for educating clients.

Treatment

Traditionally, closure of a patent urachus has been hastened by topical application of a silver nitrate-tipped swab (Fig 6.27). However,

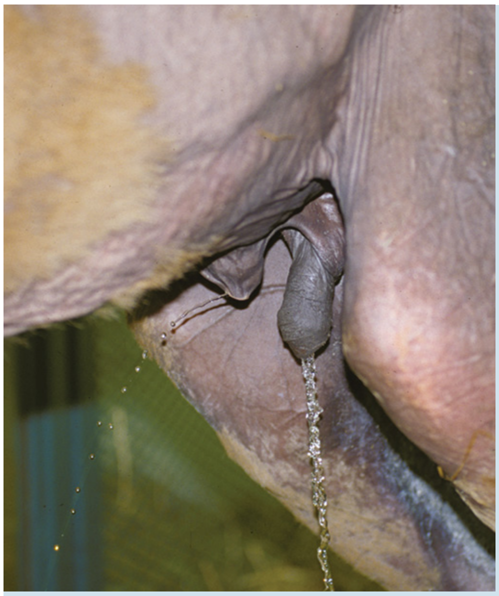


Figure 6.23: Patent urachus in a colt foal. Urine can be seen flowing from both the penis and urachus simultaneously.

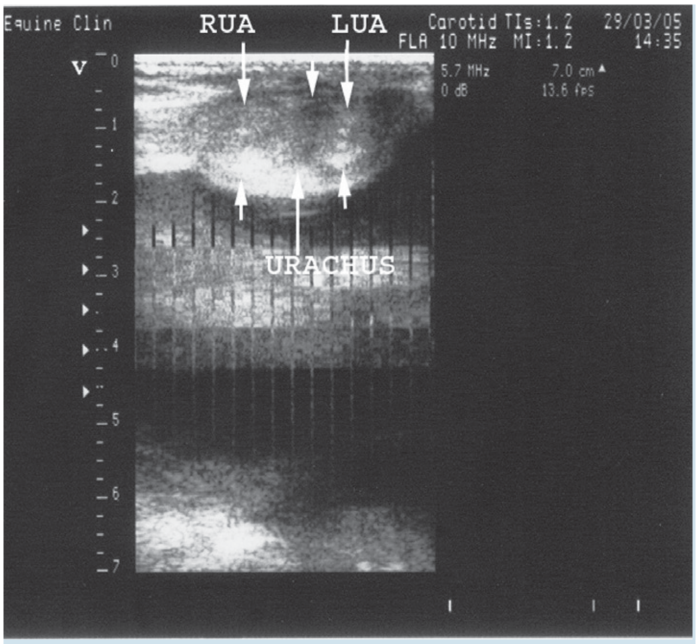


Figure 6.24: Ultrasonographic image of a normal urachus visualized between the umbilical arteries.

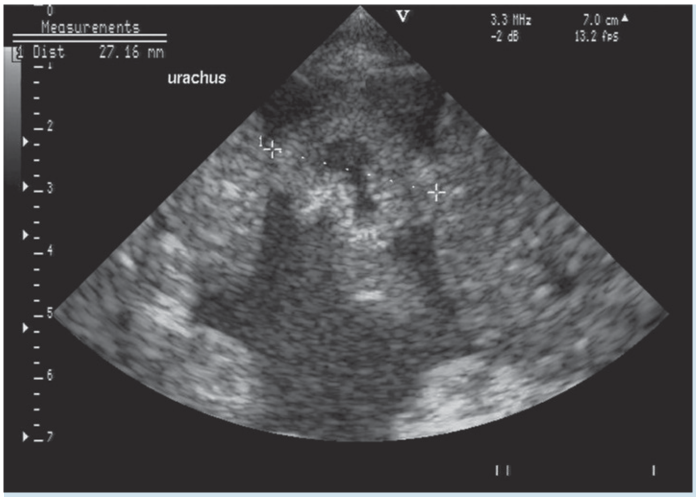


Figure 6.25: Ultrasonographic image of a fluid-filled urachus. Note also the free abdominal fluid secondary to bladder rupture.

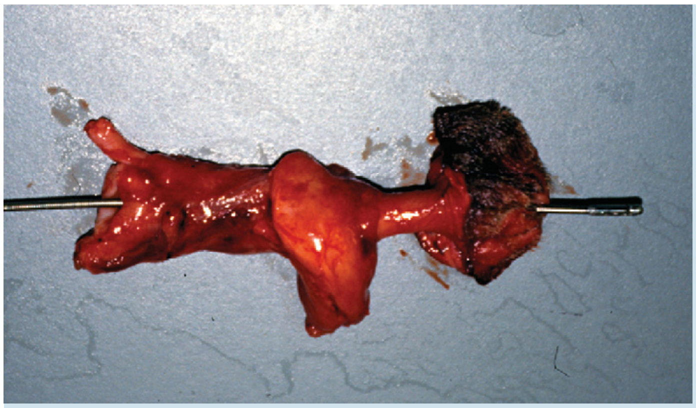


Figure 6.26: Surgically removed umbilical remnants demonstrating a patent urachus.

Figure 6.27: Application of silver nitrate to a patent urachus.

insertion of materials into the lumen of the external umbilical remnant may not be the best recommendation in all situations, and many practitioners recommend treatment consisting only of 5–7 days of antimicrobial support and keeping the foal's ventral abdomen clean and dry, in concert with twice- to four-times-daily dipping of the external umbilical stump with a 1 : 4 chlorhexidine : water or dilute iodophore solution.

Figure 6.28: Abdominal distension associated with uroperitoneum and a ruptured bladder.

Figure 6.29: Dorsal view of abdominal distension in a foal with uroperitoneum.

Acquired disorders of the urogenital tract

Uroperitoneum (Figs 6.28–6.49)

History

Uroperitoneum, or urine in the peritoneal cavity, is a common problem of neonatal foals. It is a sign of rupture of some portion of the urinary tract. The urinary bladder is the most common site of rupture, but

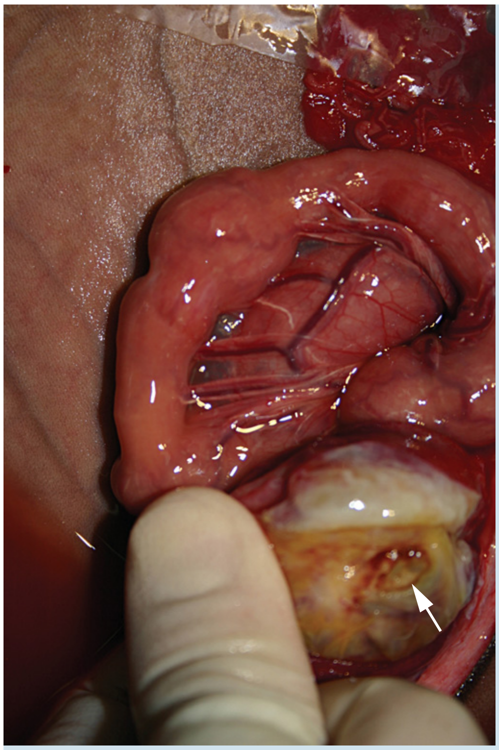


Figure 6.30: Necrosis of the bladder wall associated with an intramural abscess (arrow) in a 2-month-old foal that had previously suffered from salmonellosis.

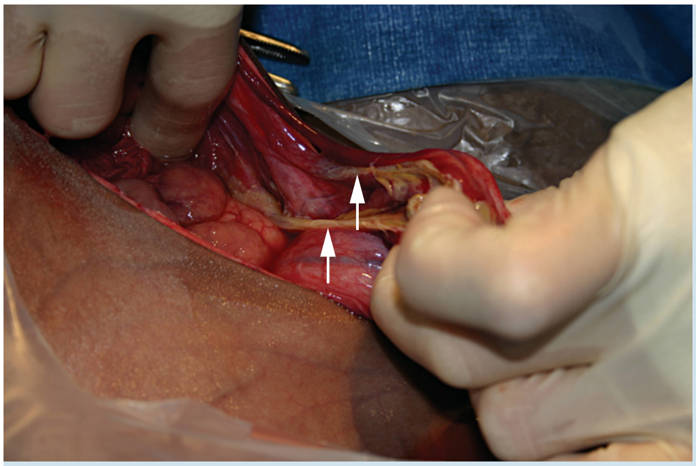


Figure 6.31: Same foal as in Fig 6.30. This image demonstrates adhesions between the bladder and the lateral abdominal wall (arrows). The adhesion and necrotic area of bladder wall were resected and the bladder repaired. The foal made an uneventful recovery.

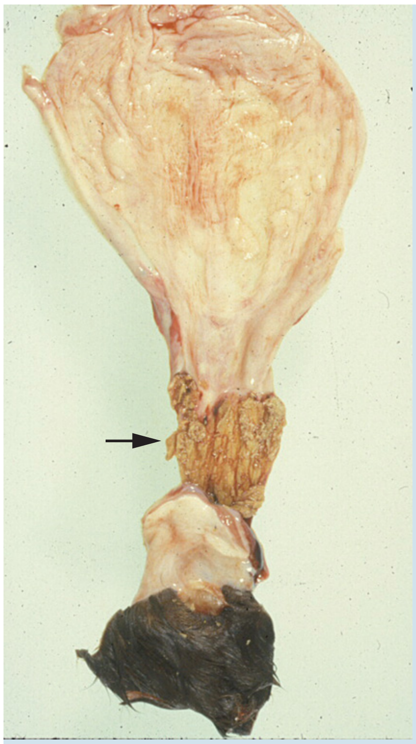
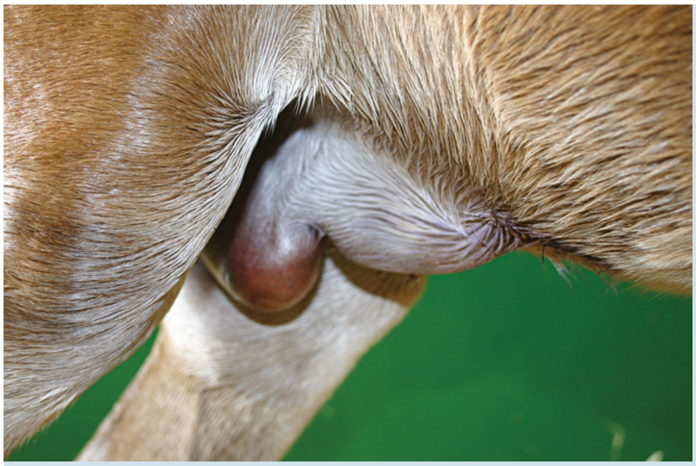


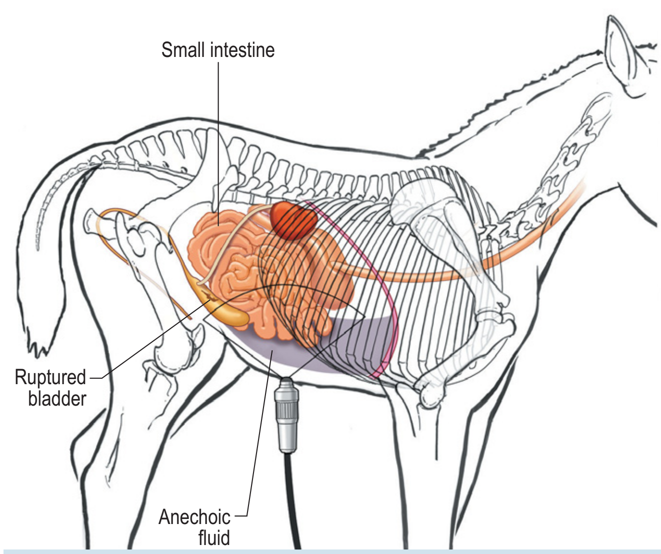
Figure 6.32: Postmortem image of a bladder and urachus with a well defined area of necrosis in the urachus (arrow) that had resulted in uroperitoneum.

Figure 6.33: Urethral tears may result in uroperitoneum or urine infiltration of scrotal tissues or both. This image is of a foal with marked scrotal swelling associated with a distal urethral tear. This foal did not have uroperitoneum. Note the tearing of the cutaneous tissue.

Figure 6.34: This foal also has marked swelling of the scrotum associated with a urethral tear which has resulted in tearing of the scrotal wall.



Figures 6.35 & 6.36: Urethral tears may also result in unilateral or bilateral enlargement of the scrotum and subcutaneous tissues of the ventral abdominal wall, depending on the site of the lesion. This 1-day-old Arabian foal had unilateral swelling. Placement of a urinary catheter for 72 hours resulted in complete resolution of the swelling with no recurrence following catheter removal.



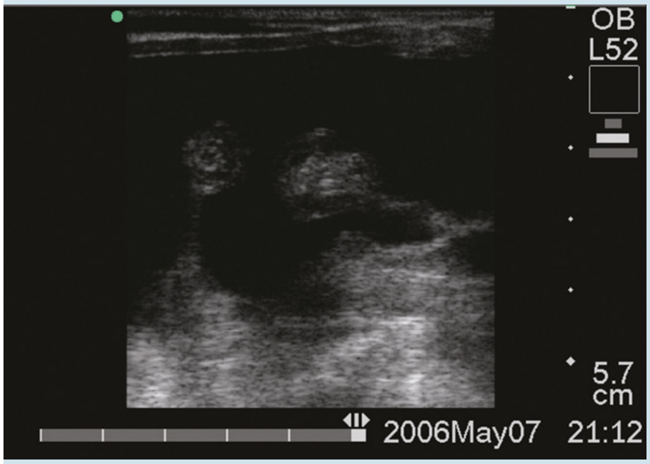
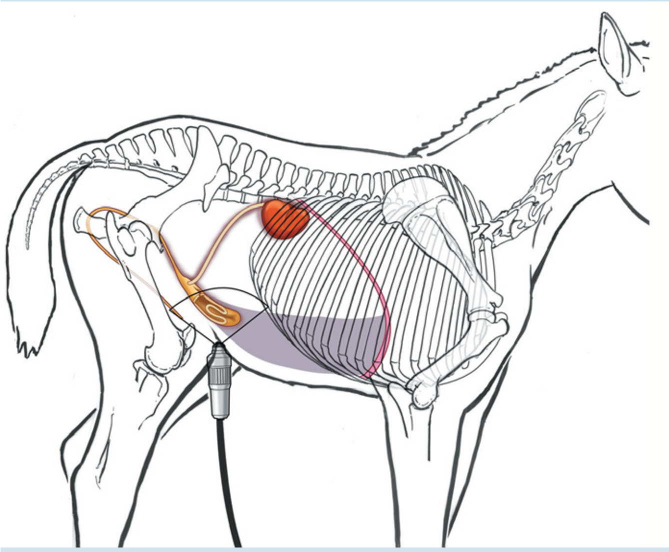


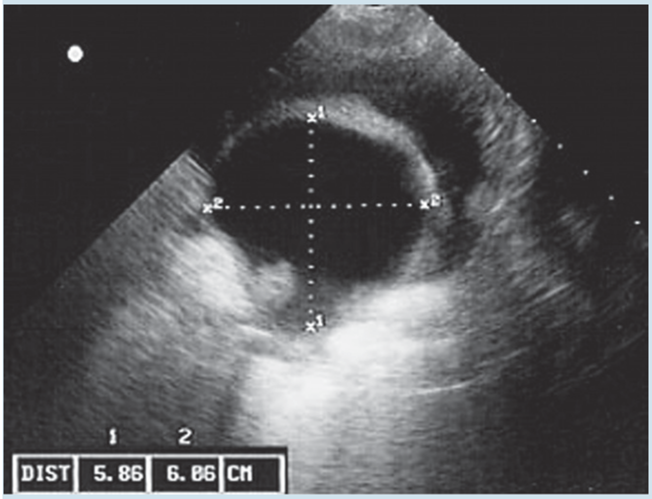
Figure 6.38: Schematic and sonographic images of uroperitoneum. Note the loops of intestine which in real time can be seen to float in the free abdominal fluid (schematic from Divers TJ, Perkins G 2003 Urinary and hepatic disorders in neonatal foals. Clinical Techniques in Equine Practice 2:67–78).

Figure 6.37: Defects in the urachus as it passes through the body wall can result in urine accumulation in subcutaneous tissues, as in this 1-day-old TB foal. Similar to urethral tears, placement of a urinary catheter for 2–3 days normally yields good results. If the urine accumulation is large or long standing, necrosis of the subcutaneous tissues can occur.

(a)



(b)



(c)

Figure 6.39: Schematic and sonographic images of the caudal abdomen demonstrating free abdominal fluid and anechoic fluid (urine) within the bladder. Depending on the size, location and duration of the defect the bladder may contain anechoic urine or be collapsed and folded upon itself (schematic image (a) & ultrasound image (c) from Divers TJ, Perkins G 2003 Urinary and hepatic disorders in neonatal foals. Clinical Techniques in Equine Practice 2:67–78).

Figure 6.40: Abdominal drain in place. Note the large volume of urine that has been already been removed from the abdomen.

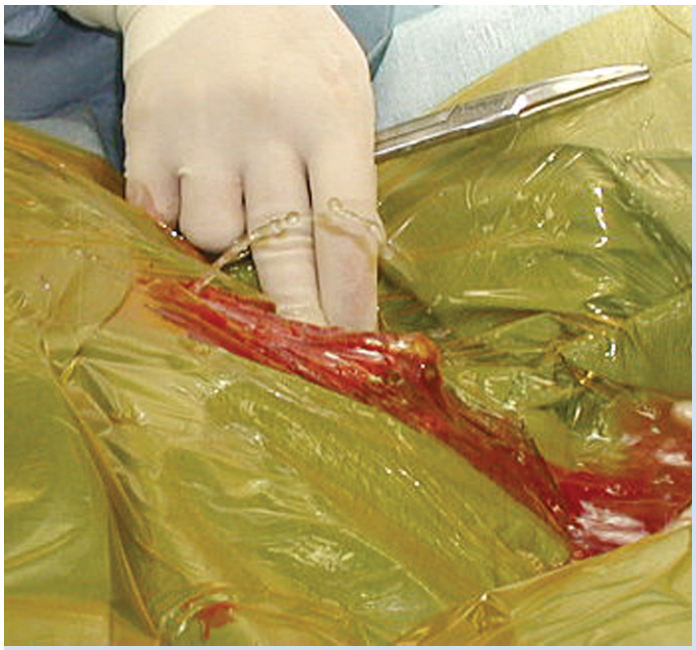
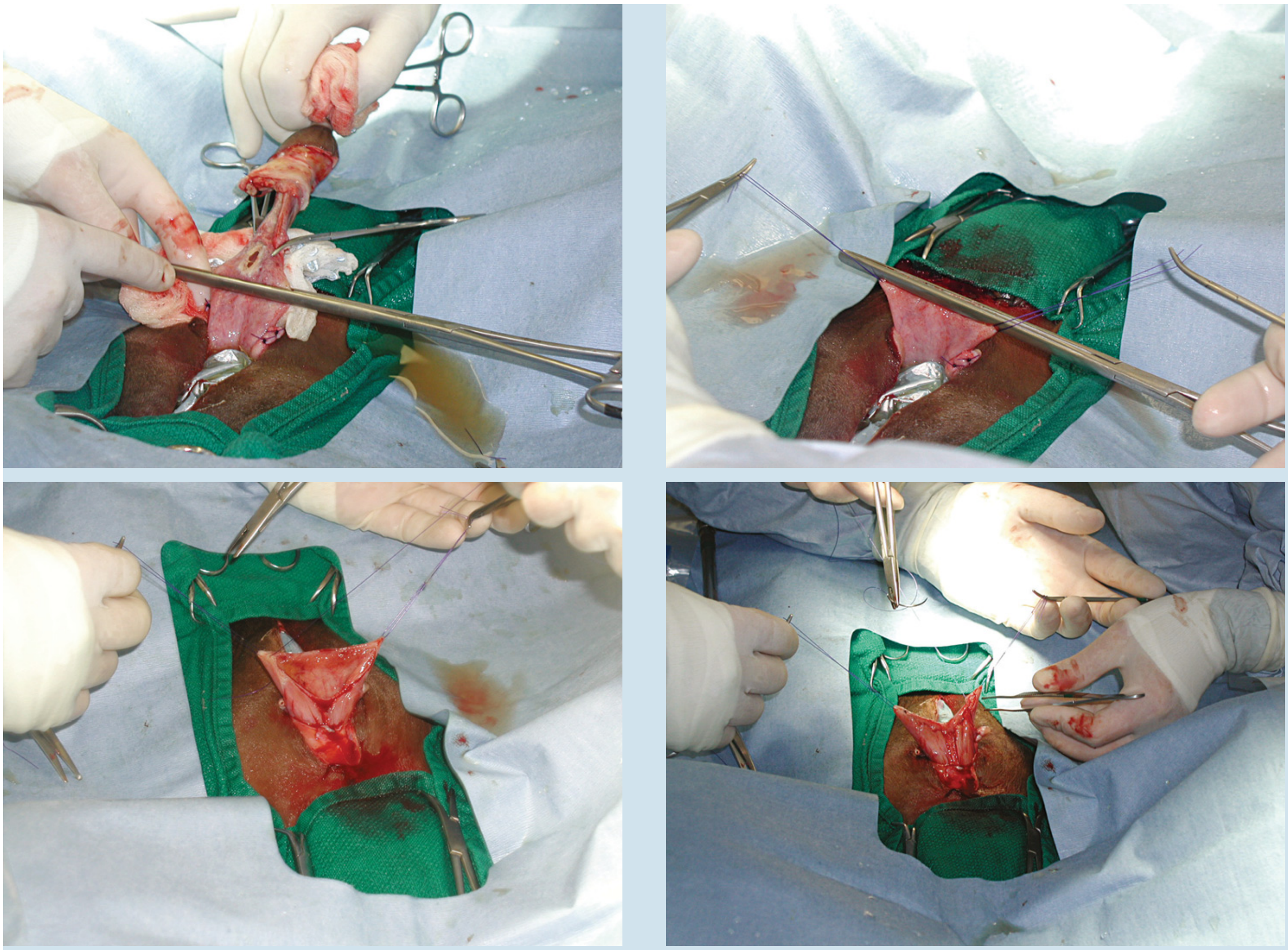


Figure 6.41: Free fluid exiting the abdomen following a ventral midline incision. In many long-standing cases it is not possible or practical to remove all the free fluid prior to surgery.



Figures 6.42–6.45: Repair of dorsal bladder tear with resection of the area containing the tear and subsequent suturing of the bladder wall.

Figure 6.46: Indwelling urinary catheter in place following surgery to repair a bladder wall defect. The condom fitted at the end of the catheter acts as a one-way valve allowing urine to flow out but preventing air from entering and inflating the bladder.

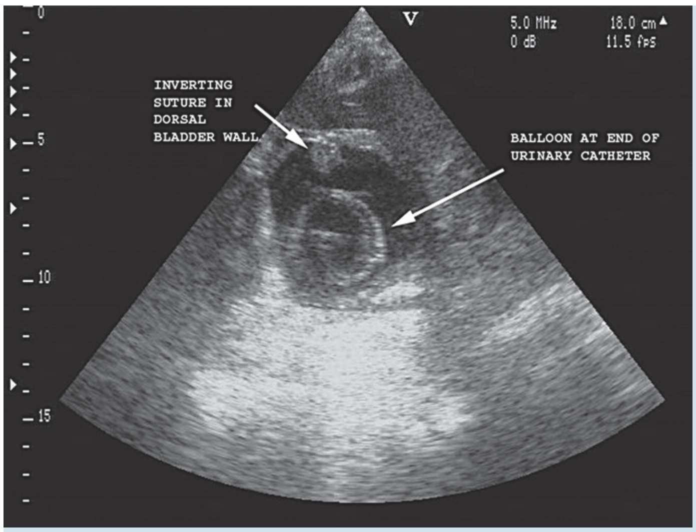


Figure 6.47: Ultrasonographic image of a urinary catheter in place following surgical repair of a bladder wall defect. Note the inverting suture of the bladder wall and the inflated cuff of the catheter.

Figure 6.48: Subcutaneous leakage of urine following an unsuccessful bladder repair.

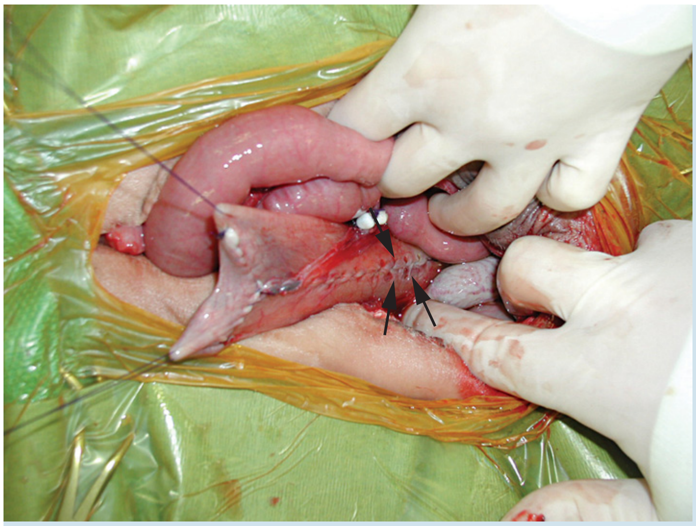


Figure 6.49: This image demonstrates necrosis (arrows) along the suture line in a previously repaired bladder. Such necrosis results in leakage of urine around the sutures and a resultant uroperitoneum.

renal, ureteral, and urethral ruptures or injuries also occur and result in leakage of urine into the peritoneal cavity.

Uroperitoneum arising from bladder rupture may occur from disruption of the urinary tract during parturition or as a result of a congenital abnormality. Bladder rupture can also result from septic foci and consequent necrosis and when caused by sepsis may be seen in older foals. Traditionally bladder rupture has been more frequently associated with male foals, but in the author's experience, the frequency with which ill or recumbent foals of either sex may be affected is approximately equal.

Hospitalized foals may not void urine voluntarily and may develop ruptured bladder after a period of hospitalization, during which time they have been receiving intravenous crystalloid fluids as supportive treatment. In such foals, the classic electrolyte imbalances may be absent or reduced in severity, although azotemia is evident.

Clinical signs

- In otherwise healthy neonates clinical signs include repeated posturing to urinate and stranguria during the first two days of life. ***The urine stream is typically weaker than normal and sporadic.***
- If the volume of urine in the peritoneal cavity is large and the rent in the bladder wall of considerable size, urine communicates freely between the peritoneal cavity and bladder lumen, and some voiding of urine via the urethra continues. Therefore, ***observation of the foal voiding urine should not rule out the possibility of ruptured bladder***.
- Stranguria can easily be misinterpreted as tenesmus making the initial diagnosis difficult.
- Urine progressively accumulates in the abdomen resulting in depression and progressive abdominal distension over 2–4 days.
- As the condition progresses these foals develop ***hyponatremia***, ***hypochloremia***, ***hyperkalemia*** and ***azotemia***. Depending on the timing of presentation, the metabolic derangements that develop can result in other signs: hyponatremia can result in neurologic disturbances including convulsions; progressive hyperkalemia results in cardiac dysrhythmias.
- Lethargy, depression and a loss of interest in nursing develops as the metabolic status deteriorates.

Differentials

- Stranguria can be misinterpreted as tenesmus seen with:
  - meconium retention
  - atresia ani
  - atresia coli.
- Bloating from gas or colonic distension (as with meconium impaction) is also common in foals, but those foals tend to have signs of colic and the abdominal distension affects the dorsal and ventral quadrants of the abdomen. In foals with uroperitoneum, signs of discomfort tend to be more vague or absent, and the abdomen appears pendulous ventrally. A fluid wave may be balloted across the abdominal wall.
- Convulsions associated with hyponatremia may resemble:
  - hypoxic–ischemic encephalopathy
  - meningitis.
- When presented in a semi-coma, the condition may resemble sepsis.
- Uroperitoneum may also be caused by disruption of the urethra, urachus or ureters (rarely). In cases of urethral leakage in colts subcutaneous accumulation of urine in the scrotal area is frequently seen. Depending on the site of injury this may occur alone or in combination with uroperitoneum. Uroperitoneum associated with urachal disruption may also be accompanied with subcutaneous leakage of urine in the umbilical area.

Diagnosis

- Transabdominal ultrasonographic is very useful in examination of foals with suspected uroperitoneum, and quickly reveals anechoic-to-hypoechoic fluid free in the peritoneal cavity, with bowel loops and other viscera floating on and in the fluid. The rent in the bladder, whether congenital (rare) or acquired, is nearly always located in the dorsal wall of the structure but is generally difficult to visualize. If the bladder-wall defect can be imaged it confers the advantage of allowing the surgeon to make estimates of difficulty, probable length of surgical and anesthetic times, and costs for owners.

- In foals for which surgery is not an option, or in foals with urethral tears, placement of an indwelling urinary catheter in combination with an indwelling peritoneal drain may facilitate sufficient removal of urine to permit healing, especially if the rent in the bladder wall is modest in size and dorsally situated. However, it should be noted that there are few reports of bladder wall defects that have healed with catheterization alone.
- Detection of voluminous anechoic-to-hypoechoic peritoneal fluid is often grounds for a presumptive diagnosis in the field.
- If ultrasound is not available, a fluid sample may be obtained via abdominocentesis and assayed for creatinine concentration. ***If the creatinine concentration in the peritoneal fluid is twice the serum concentration or higher, a diagnosis of uroperitoneum may be rendered.***
- It is important to note that serum electrolyte and creatinine concentrations may be normal in the early stages of the disorder and should not be used as a means of ruling out the condition.

Treatment

- Although the preferred management approach may vary from practice to practice, the considerations of treatment include closure of the defect, supportive care and broad-spectrum antibiotics for 3–7 days. The azotemia that is present in most cases is post-renal and the administration of aminoglycoside antibiotics is generally safe.
- In most instances slow drainage of the abdomen is initiated. Removal of urine from the abdomen helps to prevent worsening of the metabolic condition. In cases of severe abdominal distension resulting in respiratory compromise, abdominal drainage is a must.
- ***Uroperitoneum is a metabolic rather than a surgical emergency and surgery should be delayed to allow for correction of electrolyte abnormalities. Correction of hyperkalemia to a serum concentration <6 Eq/L is the most important consideration prior to general anesthesia. Administration of 1–3 L of 0.9% NaCl/5% glucose solution can be used to treat the hyperkalemia.***
- Prompt and appropriate aggressive intravenous fluid support is important because the third-space effect of uroperitoneum in the foal can be considerable given the large volume of urine typically sequestered in the peritoneal cavity at the time the diagnosis is made.
- In foals, the prognosis for full recovery, barring complications, is very good. Uroperitoneum may recur after surgery in an occasional foal as a result of ongoing leakage or further necrosis. When this occurs and the volume of fluid in the abdominal cavity is small, it can be managed conservatively by the placement of an indwelling catheter for 3–5 days. A second celiotomy may be required in some cases.
- Placement of a urinary catheter for the initial couple of days post-surgery to maintain an empty bladder is not usually required but should be used in foals where bladder rupture was deemed to be secondary to sepsis with necrosis of the bladder wall. Balloon-tipped Foley catheters with 10–35 mL of sterile saline solution in the balloon to keep it in a dependent position in the bladder lumen work well, but rigorous monitoring and nursing care, including the administration of broad-spectrum antimicrobials, is necessary. Some foals may strain with the Foley catheter and therefore are given phenazopyridine orally 4 mg/kg PO q 8 h for 2–5 days post-surgery. Phenazopyridine relieves urinary tract pain, burning, irritation and discomfort by having a direct analgesic effect on the muscosa lining the urinary tract.

Omphalophlebitis (umbilical infection) (Figs 6.50 & 6.61)

- An umbilical infection may be obvious, manifested by swelling or drainage of purulent material from the external opening, but it may also be an occult condition, with infection limited to the internal portion of the umbilical remnant.
- In the latter instance, there may be no palpable or visible abnormalities associated with the umbilicus and ultrasonographic imaging is required to detect the infection.
- Affected foals are typically febrile and have inflammatory changes on a hemogram, especially leukocytosis, neutrophilia, and high plasma fibrinogen concentration.
- In neonatal foals (i.e. less than a few days old), the alterations on a CBC may reflect Gram-negative bacterial sepsis as opposed to chronic inflammation, and fever may be variable or absent.
- In foals with pyrexia or inflammatory CBC changes that cannot be explained by external signs of infection, careful sonographic examination of the umbilicus should be performed.
- Perinatal foals that are examined because of an owner complaint of lameness should be suspected of having septic arthritis, even if the owner suspects trauma to a limb from the mare stepping on the foal. Although the latter does happen, lameness in perinatal foals is often secondary to septic arthritis. Foals in which septic arthritis is diagnosed are also candidates for prompt and careful ultrasonographic evaluation of the umbilicus.
- Ultrasonographic imaging of the umbilicus in foals is best performed with the foal in a recumbent position. Transducers with a wavelength of 7.5 MHz and a built-in stand-off yield superior images, but 5.0 MHz probes also yield images of sufficient quality to aid in diagnosis. The best images are obtained after clipping the hair from the abdomen and around the umbilical stump.
- Infections involving the external portion of the umbilicus are easily recognized. Even if drainage of purulent material can easily be established, ultrasonographic imaging may be indicated to ascertain the extent to which infection extends internally.
- In foals with internal umbilical infections, ultrasonography is invaluable not only in detecting the infection, but also for serial monitoring during treatment or after surgery.
- Enlargements in the dimension of the umbilical structures should prompt suspicion of infection, but even structures which are not enlarged in diameter may still contain fluid, exudate, or gas echoes that are suggestive of anaerobic bacterial infection, so umbilical structures should be evaluated on the basis of appearance as well as cross-sectional diameter. Involvement of multiple structures is common. Infection of the urachus and umbilical arteries is most common, with less frequent involvement of the umbilical vein.
- As a rule of thumb, the umbilical vein and each umbilical artery should not be greater than 10 mm in diameter in foals <10 days of age. Foals by 10–14 days of age should have umbilical remnants <5 mm in size. An infected umbilical vein is enlarged (>10 mm) with the most prominent enlargement at or near the external

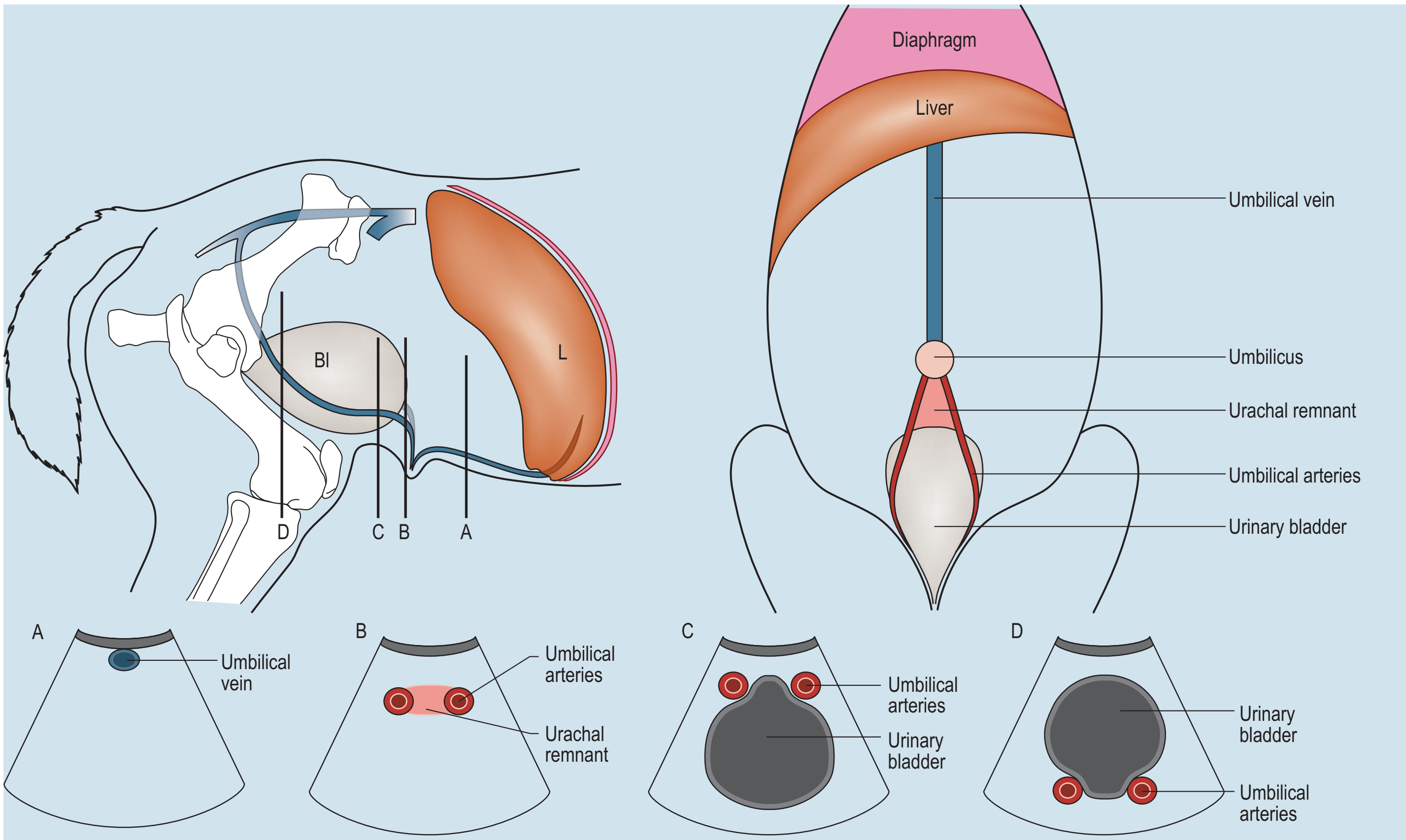


Figure 6.50: Schematic image of ultrasonographic of umbilical structures.

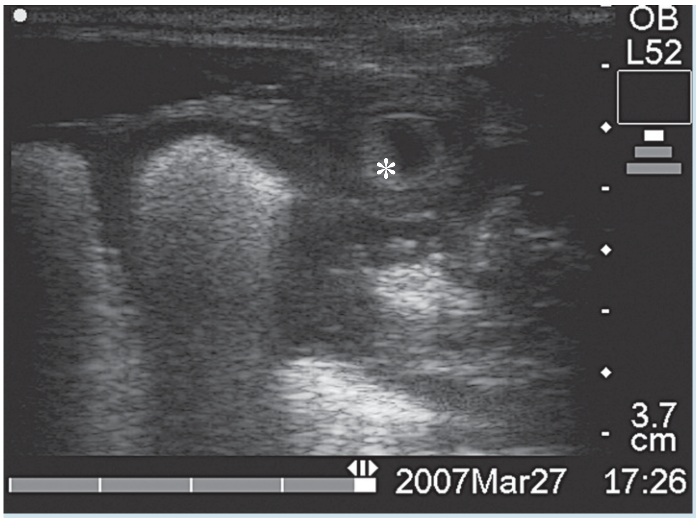


Figure 6.51: Ultrasonographic image of the umbilical vein 6 cm cranial to the umbilicus (1-day-old foal). Note the clot within the vein (*) – this is a normal finding. This image corresponds to scan plane A in Fig 6.50.

umbilical remnant. Thickening of the walls of the umbilical vein is likely with long-standing infection. Extension cranially into the liver may result in hepatic abscesses or suppurative hepatitis and warrants a thorough sonographic examination of the liver.

- ***The combined urachus and umbilical artery bundle, which courses from the bladder apex to the body wall, should not be greater than 25 mm in diameter.*** An infected umbilical artery is enlarged (>13 mm) with a thickened wall and contains anechoic to echoic fluid. Although the greatest increase in diameter of the artery may occur anywhere along its length, the infected umbilical artery is usually largest just caudal to the external umbilical remnant or at the apex of the bladder. Long-standing infections may become walled off and appear as encapsulated abscesses.
- Omphalophlebitis may successfully be treated medically (drainage, if appropriate, and antimicrobial treatment) or surgical removal of the involved structures may be necessary.
- Exudate should be submitted for bacterial culture so that antimicrobial selection can be targeted. In foals in which the infection can only be detected sonographically, broad-spectrum antimicrobial therapy is indicated.
- The prognosis for foals with umbilical infection is generally good, provided the condition is not a manifestation of Gram-negative bacterial sepsis.

Acute renal failure (Figs 6.62 & 6.63)

History

- As with adult horses, foals may develop acute renal failure in association with toxic, septic, obstructive, or hemodynamic causes.
- Foals with acute renal dysfunction typically have obvious signs of malaise.

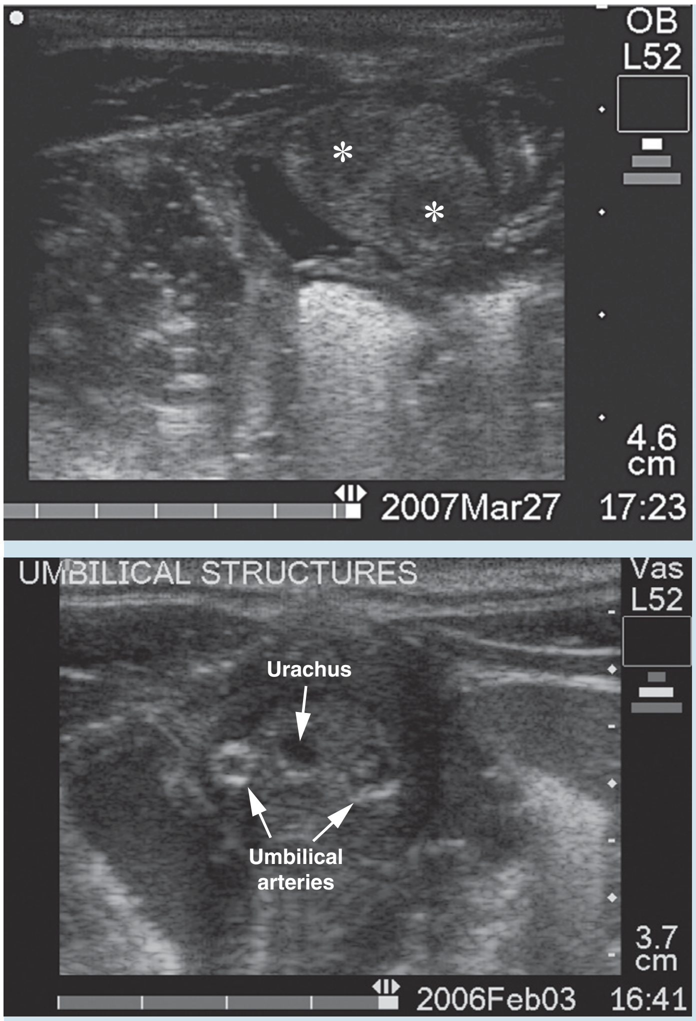


Figure 6.52: Cross-sectional ultrasound images of the umbilical structures at the level of the umbilicus in a 1-day-old foal (different foals). The umbilical arteries are marked *. These images correspond to scan plane B in Fig 6.50.

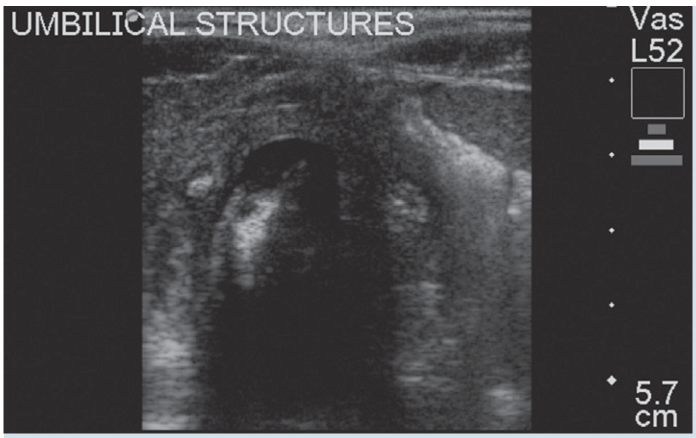


Figure 6.53: Ultrasound image of the remnants of the umbilical arteries and bladder. This image corresponds to scan plane C in Fig 6.50.

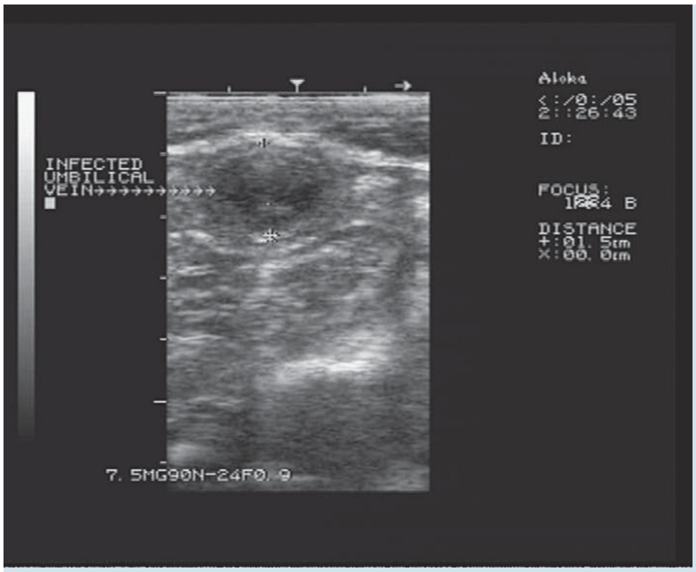
Figure 6.54: Swelling of the external umbilicus with purulent exudates.

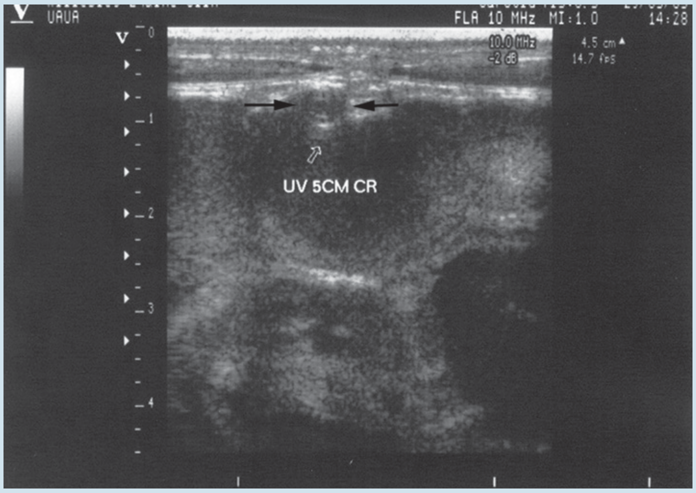
Figure 6.55: Aspiration of purulent material from a swelling of the external umbilicus.

- The management of ill foals often includes a broad-spectrum antimicrobial regimen that includes an aminoglycoside drug and NSAIDs to control pyrexia and discomfort. Renal insult is a not uncommon complication of both the septic process itself and of the treatment prescribed for it.
- Foals born with ischemic disease secondary to birth asphyxia also typically have some degree of renal insult, clinical or subclinical.
- Commonly used aminoglycoside drugs such as gentamicin and amikacin may cause tubular necrosis; signs of toxicosis are associated with prolonged treatment and excessive trough serum concentrations.
- Foals which are administered oxytetracycline as a treatment for "contracted tendons" may also have renal insult, including renal failure, because of the nephrotoxic effects of the drug.

Diagnosis

- Toxic insult results in decreased filtration, manifested clinically as oliguria.
- Biochemical analysis:





Figures 6.56 & 6.57: The image on the left is an ultrasonogram of an infected umbilical vein 5 cm cranial to the umbilicus. Note the thickened walls of the vein and the echoic fluid (purulent exudate) within the lumen of the vein. Note the thicker (15 mm) than normal (<10 mm) measurement of the vein. Compare this to an image of a normal umbilical vein at the same site in Fig 6.57. These images correspond to scan plane A in Fig 6.50.

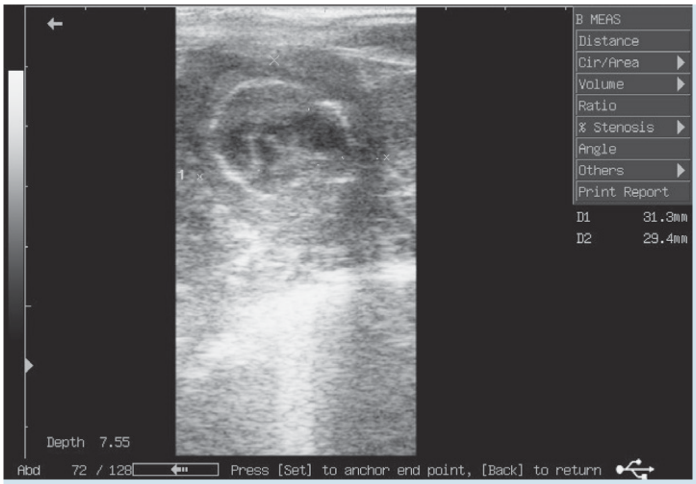


Figure 6.58: Infected umbilical vein. This 8-day-old foal had a history of fevers, elevated WCC (23,000 cells/μl) and a serum fibrinogen of 1000 mg/dL. This image was taken 5 cm cranial to the umbilicus, corresponding to scan plane A in Fig 6.50.

Figure 6.59: An infected umbilicus prior to resection. Note the hard swelling around the umbilicus that does not reduce when the foal is placed in dorsal recumbency.

- early signs of renal insult can be detected via urinalysis, in which protein and tubular casts which are not normally detected may become apparent
- decreased filtration results in increased serum drug concentrations and progressive azotemia.

- Ultrasonographic examination:
  - in addition to changes on a serum biochemical analysis, ultrasonographic examination of the foal's kidneys may also be useful
  - renal cysts, solitary or in multiples, can easily be appreciated ultrasonographically
  - there are no specific anatomic alterations in kidneys of foals with acute renal dysfunction secondary to hypoxic insult from birth asphyxia, but the organs may appear engorged or swollen and more sonolucent than normal; in some instances anechoic perirenal fluid accumulation may be apparent.
  - kidneys of foals with toxic changes of drug-induced nephrotoxicosis may appear more echogenic than normal, consistent with focal or diffuse parenchymal infiltration; echogenic debris in the renal pelvis has been detected in foals with suspected NSAIDs toxicosis.

Treatment of acute renal failure in foals

Monitoring urine production

- The ability to verify that urine is being produced and quantifying the volume produced are necessary for proper monitoring of the oliguric or anuric foal.

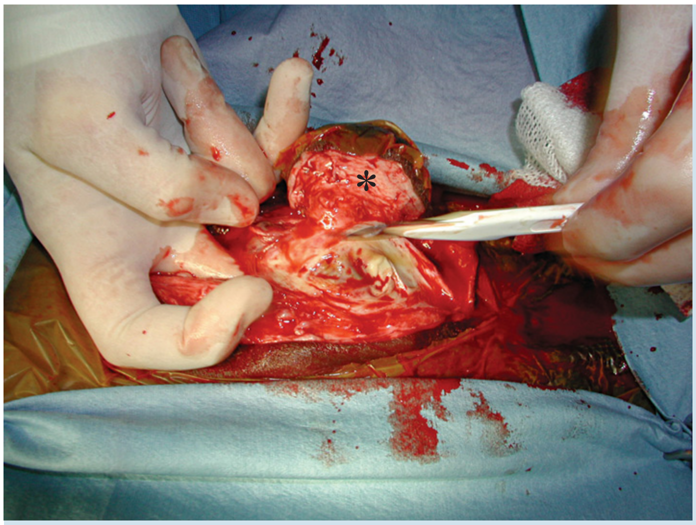


Figure 6.60: Resection of the umbilicus shown above; note the fibrotic tissue surrounding the umbilicus (*).

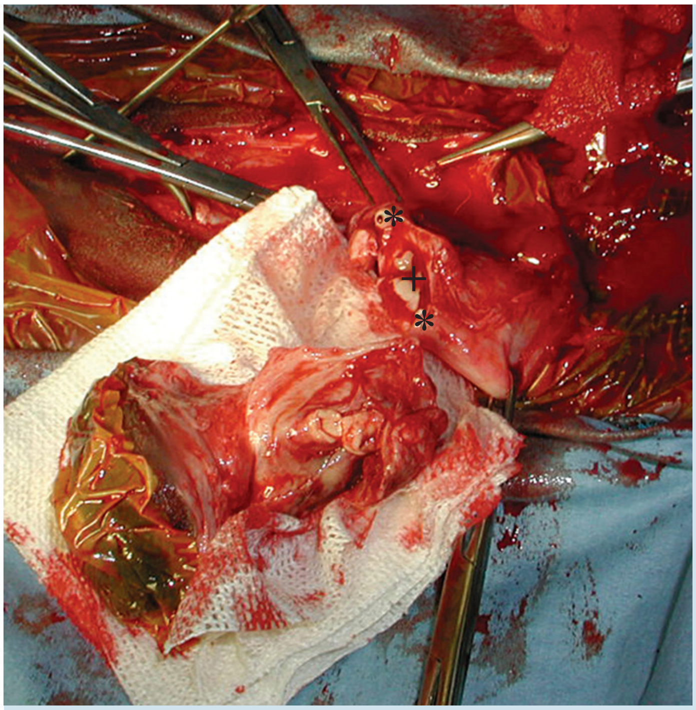


Figure 6.61: Separation of the umbilical remnants at the bladder. Note the remnants of the umbilical arteries (*) and the bladder (+).

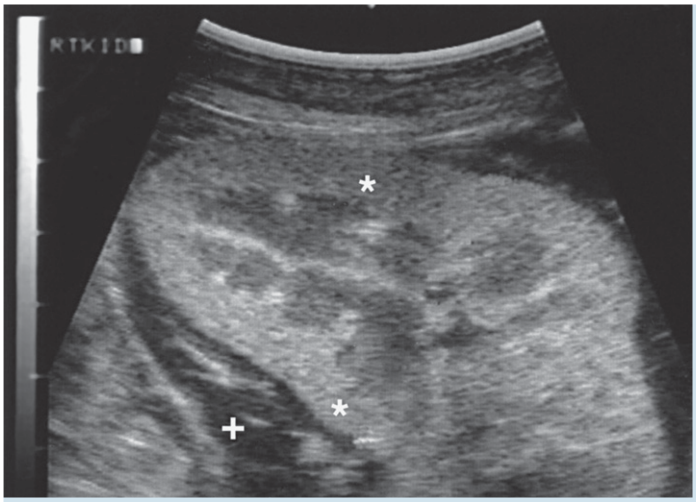


Figure 6.62: Ultrasonographic image from a 6-day-old foal with ARF. The cortex (*) appears brighter than normal, at least partially attributable to decreased perfusion, and there is perirenal edema (+). The perirenal edema is a guarded to poor prognostic finding with acute intrinsic renal failure (ultrasound scan Divers TJ, Perkins G 2003 Urinary and hepatic disorders in neonatal foals. Clinical Techniques in Equine Practice 2:67–78).

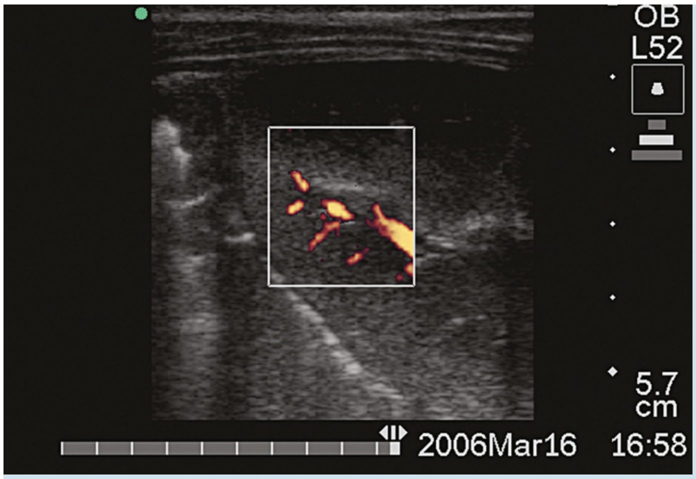


Figure 6.63: Sonographic image demonstrating adequate blood flow in a foal with azotemia.

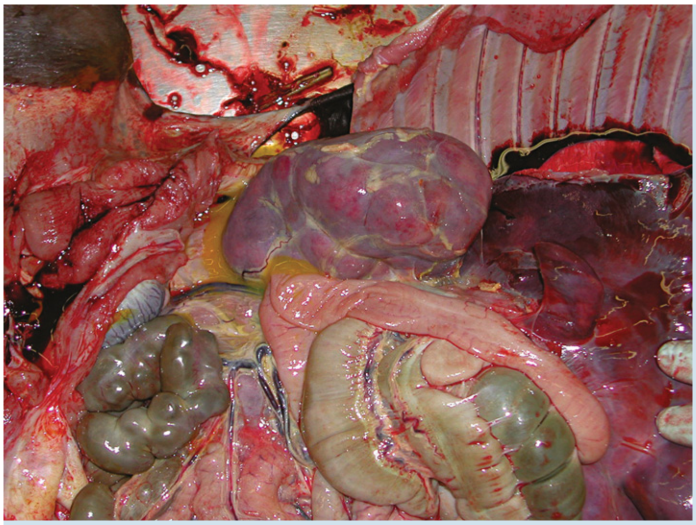
- Placement of an indwelling urinary catheter is necessary. The placement procedure should be performed aseptically because the indwelling collection system represents a non-physiologic portal of entry and can lead to added infectious burden in an already-ill foal (see Chapter 3, p. 72).
- Recumbent foals are also more likely to experience bladder rupture; therefore, given that administration of intravenous fluids and diuretic pharmaceuticals form the basis for treatment of foals with renal insult or failure, catheterization to facilitate monitoring of urine output is essential.
- Proper placement of the catheter should be verified with transabdominal ultrasonographic imaging; the fluid-tipped catheter can be clearly imaged in situ (see Fig 6.47). It is not unusual for indwelling catheters to be expelled from the bladder, even if sutured to the preputial or vulvar skin, particularly if the foal is ambulatory, necessitating re-placement.

Promoting diuresis with intravenous fluids

- Diuresis is usually promoted by administration of intravenous fluids and diuretics. However, it is important to establish whether the renal failure is oliguric or polyuric in nature because inappropriate infusion of IV fluids can quickly lead to increased central venous pressure and pulmonary edema in foals with decreased filtration. The loop diuretic furosemide and the osmotic agent mannitol have both been used to promote diuresis in foals with renal failure; dopamine also has diuretic effects, but its use should be limited to

situations where blood pressure can be monitored if the patient is oliguric.

- If initial volume replacement with crystalloid fluids, with or without colloids, does not elicit urine production soon after indices of rehydration are noted (e.g. improvement in skin turgor, improvement in mucous membrane perfusion, improvement in enopthalmia, decrease in severity of tachypnea, improved jugular refill, increased alertness), it is advisable to continue fluid therapy conservatively.
- Monitoring CVP is important, because in foals that are oliguric or anuric, volume overload develops rapidly in the absence of filtration of plasma by the kidneys, resulting in pulmonary edema as well as edema formation in various capillary beds throughout the body. Central venous pressure can be monitored with reasonable ease even in the field once an appropriate indwelling IV catheter has been placed (see Chapter 3, p. 68). CVP should be maintained in the range of 10–12 cm H_2O, but knowledge of the baseline CVP and trends in the CVP are important.
- Guidelines for formulating replacement and maintenance fluid administration plans vary, and it must be recognized that no one formula is likely to be optimum for all foals. A plan that is useful in many instances is to administer a series of fluid boluses of 10–20 mL/kg, each (of a crystalloid solution) given over a 10–30-minute interval. The foal should be monitored during and after the completion of each fluid bolus to determine if there are improvements in hemodynamic parameters and whether additional volume is needed. Colloidal fluids may be administered concurrently with, or instead of, the crystalloid fluids, at a dose of 3–10 mL/kg. Published recommendations for maintenance fluid requirements are likewise varied; one widely used rule-of-thumb calculates that maintenance volume requirements are 80–120 mL/kg/day. Another formula, which may more accurately approximate the foal's needs, is to administer *100 mL/kg/day for the first 10 kg of body weight +50 mL/kg for the second 10 kg of body weight, and 20–25 mL/kg/day for the remaining body weight*.
- In foals with oliguric or anuric renal failure, all formulas must be balanced against alterations in CVP and manifestations of edema. Once volume replacement has been achieved and CVP is in or near reference range (10–12 cm H_2O), if urine output is absent or minimal, administration of diuretics should be initiated.
- Concurrent administration of dobutamine (5–10 μg/kg/min) and dopamine (1–5 μg/kg/min) as an IV infusion has also been used with anecdotal success in many foals. One vial of dobutamine (250 mg) and one vial of dopamine (200 mg) in 500 mL saline given at 0.45 mL/kg/h will calculate to 3 μg/kg/min of dopamine and 3.6 μg/kg/min dobutamine.
- Aminophylline may inhibit renal vasoconstriction and has mild diuretic effects (5 mg/kg IV given as a slow infusion twice daily).
- Mannitol (0.5–1.0 g/kg, given IV over 1–2 hours) may be given to support continued diuresis once urine formation commences.
- If medical treatment is unsuccessful at establishing diuresis, peritoneal dialysis may be performed.
- Ultrasonographic imaging of the kidneys and bladder can be helpful at informing the examiner whether renal blood flow appears adequate and whether urine is accumulating in the bladder (or peritoneal cavity). Assessment of renal blood flow as an indication of acute renal failure in foals is becoming more commonplace but to date there has been no controlled study of its prognostic value. Children with acute renal failure have an absence of blood flow in late diastole or throughout diastole that is easily detected with Doppler ultrasound.



Figures 6.64 & 6.65: Postmortem samples of a foal that was suffering from leptospirosis: note the haemorrhages evident in the kidney in situ and in cross-section.

Pyelonephritis (Figs 6.64–6.66)

Pyelonephritis in foals is uncommon but when it is diagnosed, it usually has developed via the route of hematogenous seeding as a complication of bacterial sepsis. Bacterial culture of urine or postmortem renal biopsy specimens in young foals has yielded growths of *Actinobacillus* spp, *Salmonella* spp, *E. coli* spp, and *Streptococcus* spp at one private referral center. Leptospirosis is also a cause of nephritis and pyuria.

Clinical signs

- Clinical signs in affected foals include fever, depression, and weight loss.
- Alterations in clinical pathology values typically include leukopenia or leukocytosis (depending on the stage of disease at which blood is collected for analysis; the latter finding is more frequent), hyperfibrinogenemia, hyperglobulinemia, azotemia, and derangements involving acid–base and electrolyte balance if both kidneys are involved.

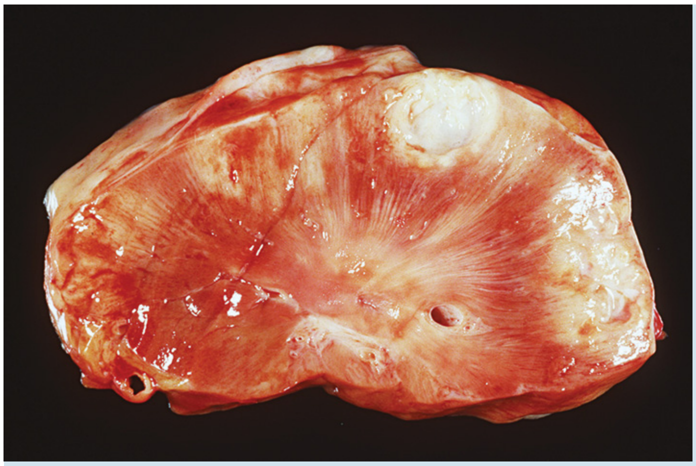


Figure 6.66: Pyelonephritis. Note the large abscesses obliterating much of the renal parenchyma.

- In instances in which infection of the upper urinary tract has resulted from ascension of infection from the bladder (cystitis), foals may be observed straining to urinate (stranguria) or voiding inappropriately small volumes frequently (pollakiuria).
- Foals with primary upper urinary tract infection are less likely to have clinical signs of dysuria, stranguria, or pollakiuria but are more likely to have signs of serious systemic illness than foals with uncomplicated cystitis.
- In foals with either etiology of pyelonephritis, hematuria may be appreciable grossly or on a microscopic basis during cytologic examination of a urine sample, and pyuria and proteinuria are typically also detected.
- If infection with *Leptospira interrogans pomona* is suspected, a sample of urine should be obtained for culture and for antigen assay with fluorescent antibody, serology for leptospirosis should also be performed with a rising titer or a single high MAT titer (1 : 6400–1 : 819,200) being regarded as diagnostic.
- In pediatric human patients with pyuria, ultrasonographic findings of localized hyperechoic changes in the kidney (as opposed to diffuse alterations in echogenicity or accumulation of echogenic material in the calyces and pelvis) are termed acute bacterial lobar nephronia. This condition corresponds with bacterial infection of renal parenchyma and tissue of the collecting system but the term is used to describe the condition before the infection has become organized into a renal abscess. The etiology of nephronia is typically hematogenous seeding of the kidneys with *E. coli* spp as a complication of sepsis.
- Detection of changes in echogenicity in the kidneys of foals with supportive clinical signs constitutes grounds for aseptic (urinary catheter) collection of a urine sample and bacterial culture and susceptibility testing. In affected foals, diagnosis of pyelonephritis (Fig 6.65) should prompt thorough scrutiny of other internal visceral systems for similar signs of bacterial seeding given the likelihood that a bacteremic or septicemic episode likely preceded the condition.

Diagnosis

- Diagnosis of pyelonephritis is made on the basis of laboratory findings on blood and urine analysis and ultrasonographic imaging of the kidneys.
- Endoscopy of the bladder may be used in fillies to visualize the ureteral orifices and determine whether hematuria or frank pyuria, if present, is originating from one or both kidneys.

Treatment

- Renal infections are serious and life-threatening ailments. Treatment should be prescribed on the basis of bacterial culture and susceptibility testing and typically involves broad-spectrum antimicrobials, with other treatments, such as intravenous fluid support, antipyretics, and analgesics, prescribed as needed. Because some antimicrobial drugs that might be indicated as appropriate on the susceptibility test results may be nephrotoxic, it is important to weigh pharmaceutical choices carefully, especially in multiple-drug treatment regimens which may also include NSAIDs. Initial treatment with drugs that are administered parenterally may be followed by antimicrobials that have high lipid- or tissue-solubility and can be administered orally, for long-term treatment.
- Penicillin (20,000–40,000 iu/kg) is the treatment of choice for leptospirosis and should be instituted immediately if leptospirosis is suspected pending results of culture and other tests.
- The foal's response to treatment may be monitored sonographically and with serial blood and urine laboratory work. The prognosis for recovery in foals battling other complications of sepsis should be considered guarded.
- Renal scarring is a reported sequela to acute lobar nephronia or renal abscess in children, but no studies have been published in which this phenomenon was investigated in foals.

Cystitis

Cystitis is most commonly seen in foals secondary to antimicrobial treatment for bacterial sepsis, especially in foals that are recumbent. Infection with *Candida* spp is not uncommon in such foals; diagnosis is made on the basis of urine microbial culture (a request for fungal culture must be specified) or cytologic evaluation of voided urine and detection of fungal elements. Urine may be blood-tinged and may contain white flocculent precipitates. Treatment should be initiated with ketoconazole or fluconazole.

Recommended reading

Andrews FM, Rosol TJ, Kohn CW et al 1986 Bilateral renal hypoplasia in four young horses. Journal of the American Veterinary Medical Association 189:209–212

Gull T, Schmitz DG, Bahr A et al 2001 Renal hypoplasia and dysplasia in an American miniature foal. Veterinary Record 149:199–203

Graves EA 2003 Congenital disorders of the urinary tract. Current Therapy in Equine Medicine 5:826–828

Kablack KA, Embertson RM, Bernard WV et al 2000 Uroperitoneum in the hospitalized equine neonate: retrospective study of 3 cases, 1988–1997. Equine Veterinary Journal 32:505–508

Lavoie JP, Harnagal SH 1988 Nonsurgical management of ruptured bladder in a critically ill foal. Journal of the American Veterinary Medical Association 192:1577–1580

Rantanen NW 1986 Diseases of the kidneys. Veterinary Clinics of North America (Equine Practice) 2:89–103

Wong SN, Lo RN, Yu EC 1989 Renal blood flow pattern by non invasive Doppler ultrasound in normal children and acute renal failure patients. Journal of Ultrasound in Medicine 8:135–141

CHAPTER 7

Cardiovascular system

Edward Voss DVM, Dipl. ACVIM

Cardiovascular examination (Figs 7.1 & 7.2)

Evaluation of the cardiovascular system is an important part of every physical examination. The basic techniques of auscultation, palpation and assessment of mucous membrane color and capillary refill times have been covered in Chapter 3. In addition to these basic techniques there are other procedures which can be performed to aid in assessment of the function of the cardiovascular system.

Thoracic radiography

The small size of neonatal foals permits the use of thoracic radiography which has size-related limitations in older equids. Lateral and ventrodorsal images may be taken but in some situations the sedation required may in fact prohibit such positioning. Thoracic radiography can be used to identify areas of pulmonary or pleural disease and assist in ruling out differentials for cardiac disease. Increases in cardiac size and changes in shape may be detected in foals with significant cardiomegaly. Changes in individual chambers may be more difficult or impossible to detect on radiographs. Considerable experience is also required for detecting subtle lesions.

Laboratory studies

Systemic or metabolic disorders may result in cardiovascular disorders and conversely cardiovascular disorders may alter routine laboratory tests. The laboratory studies that may be used for assessing and managing cases of cardiovascular disease are outlined below. The tests chosen will depend on the suspected or confirmed condition and the treatment protocol.

- Complete blood count and fibrinogen to detect anemia and/or infection.
- Serum biochemical tests including electrolytes, renal function tests and cardiac muscle enzymes. These are useful for assessing arrhythmias, low cardiac output resulting in azotemia and myocardial injury. Cardiac muscle enzymes that are evaluated are isoenzymes of creatine kinase, lactate dehydrogenase and cardiac troponin I. Values of cardiac troponin I ≥ 1 ng/mL are highly suggestive of myocardial disease/inflammation.
- Serum protein can be used to assess hypoalbuminemia and hyperglobulinemia.
- Arterial blood gas and pH can be used to assess cardiopulmonary and renal function in addition to acid–base assessments.
- Urinalysis may be used to assess secondary renal injury.
- Blood cultures can be used for the identification of bacteremia and aid in the diagnosis of endocarditis.
- Serum or plasma assays for therapeutic drugs.
- Clotting profiles (see p. 203).

Bone marrow biopsy/aspirate (Figs 7.3 & 7.4)

Bone marrow biopsies are rarely performed but in some cases e.g. poorly responsive thrombocytopenia or Fell pony syndrome (see

Figure 7.1: Ecchymotic hemorrhages on the pinna found during physical examination indicate a disorder of the cardiovascular system and warrant further diagnostics – e.g. clotting profile.

Figure 7.2: Cardiac auscultation in a 12-hour-old foal.

Chapter 11 p. 295), they may provide invaluable information. The cortical bone of foals is obviously softer than that of an adult but the medullary cavities are also correspondingly smaller. The wing of the ileum is the most commonly used site in foals. Other sites are the proximal cranial portion of the humerus, sternum and rib. Thoracic penetration is a risk with the rib and sternum sites.

Procedure

- The foal is restrained and sedated (if required).
- The site should be aseptically prepared. If using the wing of the ileum the site is the most external point of the ileum and the needle is directed down the shaft.
- A small bleb of local anesthesic can be placed at the site.

Figures 7.3 & 7.4: Bone marrow biopsy being performed from the wing of ileum in a foal with thrombocytopenia.

- A stab incision is made with a scalpel blade.
- If using the wing of the ileum a 3 $^1/_2$ inch, 18G biopsy needle is used. The needle is introduced with firm pressure into the medullary cavity.
- If obtaining an aspirate the stylet is removed and a syringe is attached to the cannula. Brief and firm aspirations are made until a sample is obtained. The sample obtained should be placed in egg albumin EDTA unless otherwise requested by the laboratory. Smears will then be made from the sample and should be interpreted by a pathologist.
- If a biopsy sample is required: once the marrow has been penetrated the cannula should be advanced slowly without the stylet in place and then removed. The specimen can then be gently pushed out of the cannula into a receiving container. The transport medium should be discussed with the lab prior to collection.

Electrocardiogram (Figs 7.5 & 7.6)

The electrocardiogram (ECG) is used to detect cardiac arrhythmias in horses. Unlike small animals, ECGs cannot be reliably used to

Figure 7.5: Direct placement of portable ECG unit on the thorax at the level of the heart.

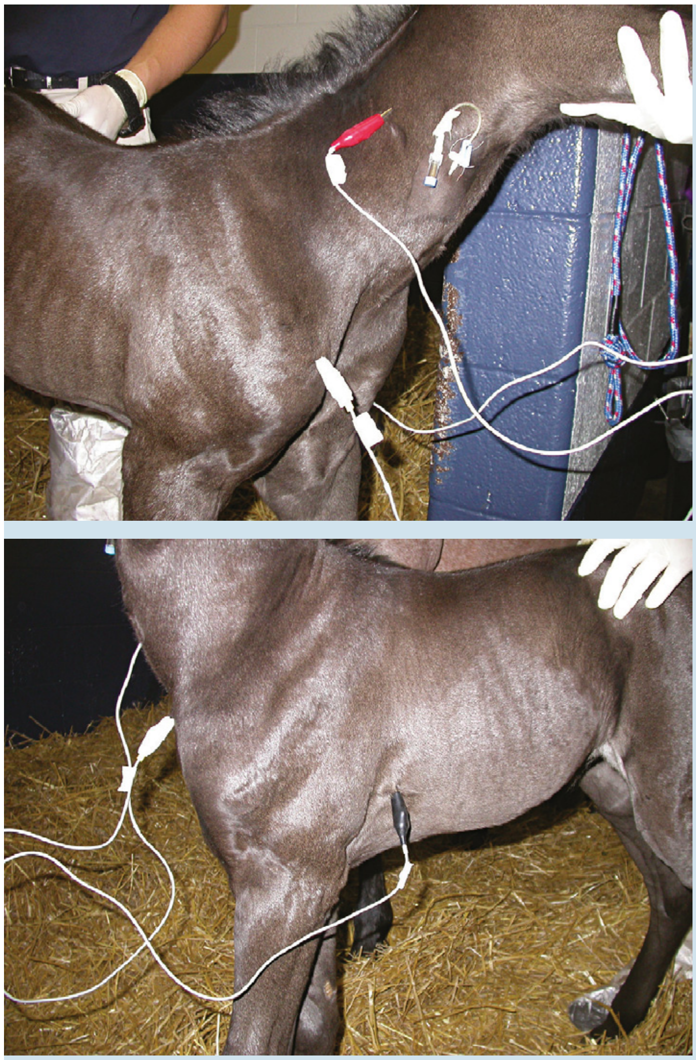


Figure 7.6: Obtaining a base-apex system ECG. (1) The right arm electrode or negative electrode (usually coded white) is clipped to the skin over the right shoulder, in the right jugular furrow or at the top of the scapular spine. (2) The left arm electrode or positive electrode (usually coded black) is placed over the apex of the left heart slightly above the line of the elbow in the "girth" region. (3) The left leg or ground electrode (red) serves as a ground and is placed at any point remote from the heart usually on the right side of the neck in the cervical region or over the "saddle" area. (4) Run the ECG on Lead 1.

detect chamber enlargement. Recording of the ECG should occur in a quiet dry place – ideally in the horse's stall where it will be most relaxed.

A number of different lead systems have been described with the base-apex lead being one of the most commonly used. Portable electrogram recording devices are also available and can be applied directly to the chest wall over the heart following the liberal application of alcohol at the site.

Echocardiography (Figs 7.7–7.14)

Echocardiography is the diagnostic tool of choice for assessing the significance of cardiac murmurs in horses of all ages. Their small size, proximity of the heart to the chest wall and relative absence of fat makes echocardiography of foals a much easier task than in adults. Standard examinations are performed from the right parasternal window with high frequency transducers (5–10 MHz) being used. Performing an echocardiogram in the field may allow for confirmation of a presumptive diagnosis; however the long-term prognosis for athletic performance may need a more detailed echocardiographic examination to be performed at a referral institution.

Cardiovascular system of the neonate (Figs 7.15 & 7.16)

The transition from the circulatory system of the placental-dependent equine fetus to the pulmonary-dependent neonatal cardiovascular orientation is complex. The primary cardiovascular function of both orientations is the same – deliver oxygen to organ systems, return deoxygenated blood to a gas exchange organ and eliminate carbon dioxide so that normal metabolic activities can continue. In order for this to occur in-utero, a number of intra-cardiac (foramen ovale), and extra-cardiac (ductus venosus, ductus arteriosus) shunts supply deoxygenated blood to the placenta and return oxygenated blood to the organs.

At parturition, gas exchange is shifted from the placenta to the lungs. This transition from fetus to neonate thus includes elimination of the placental circulatory pathways (i.e. closure of the foramen ovale, ductus arteriosus and ductus venosus), pulmonary alveolar expansion and an increase in pulmonary blood flow that accommodates the entire cardiac output of the right ventricle.

Prior to parturition, most inferior vena caval oxygenated blood entering the right atrium passes through the foramen ovale into the left atrium. At parturition, pulmonary blood flow increases and the pulmonary venous return to the left heart raises pressure in the left atrium. This left pressure gradient closes the foramen ovale by compressing the septum primum and septum secundum.

With reduced pulmonary vascular resistance, pulmonary arteriolar pressure falls below systemic vascular pressure and flow through the ductus arteriosus (DA) becomes minimal. Closure of the ductus arteriosus is additionally driven by oxygen tension and pulmonary cytokine induction (i.e. rise in bradykinin, and decreased PGE_2 and/or prostacyclin). Blood with PaO_2 >50 mmHg passing through the DA stimulates constriction of the DA wall.

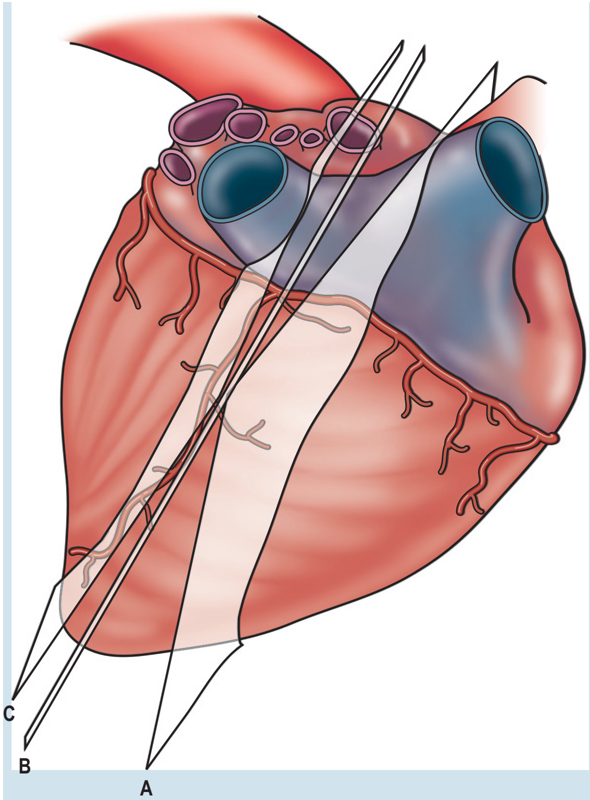


Figure 7.7: Schematic representation of the scan planes of the long axis views obtained from the right cardiac window with the probe positioned in the 4th intercostal space at the one o'clock position. (A) Scan plane of the long axis view of the right ventricular inflow and outflow tracts. The transducer is positioned in the 4th intercostal space at the one o'clock position and aimed cranially towards the opposite shoulder. (B) Scan plane of the long axis view of the left ventricular outflow tract. The transducer is positioned in the 4th intercostal space at the one o'clock position, perpendicular to the chest wall and aimed directly across the thorax. (C) Scan plane of the long axis 4 chamber view. The transducer is positioned in the 4th intercostal space at the one o'clock position. It is important to have the right front limb placed forward while aiming the transducer caudally towards the 5th intercostal space while pushing into the triceps muscle.

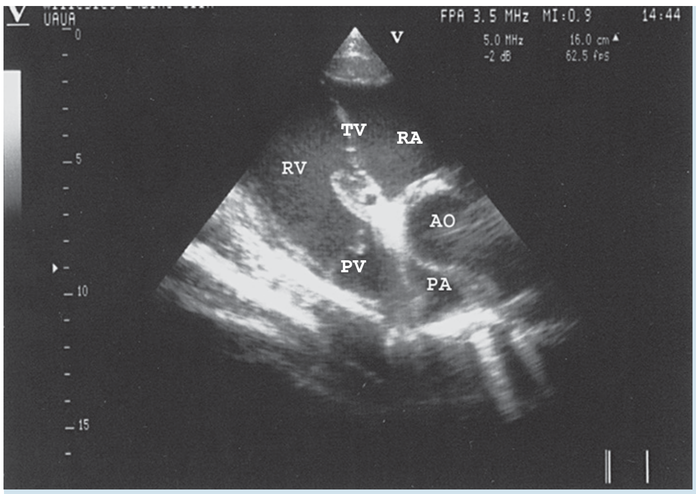


Figure 7.8: Ultrasonographic image of the right parasternal long axis view of the right ventricular outflow tract. This image corresponds to scan plane A in Fig 7.7. Structures seen with this view include: tricuspid value (TV), right atrium (RA), right ventricle (RV), pulmonic valve (PV), aorta (AO) and pulmonary artery (PA).

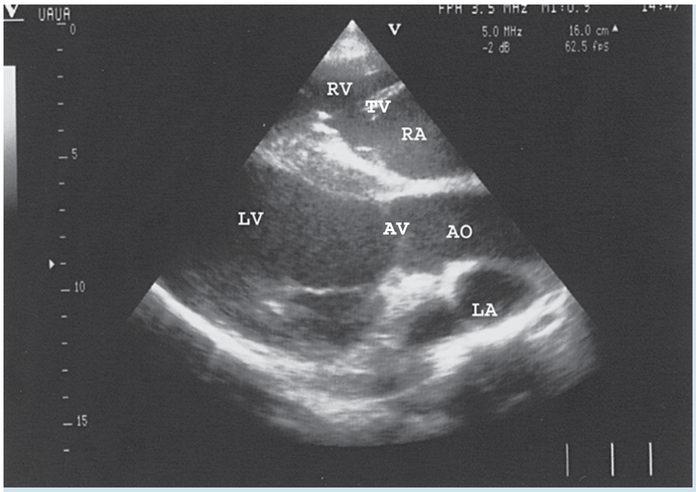


Figure 7.9: Ultrasonographic image of the right parasternal long axis view of the left ventricular outflow tract. This image corresponds to scan plane B in Fig 7.7. Structures seen with this view include: aortic valve (AV), aorta (AO), left ventricle (LV), right atrium (RA), right ventricle (RV) and the tricuspid valve (TV). This is the view in which a ventricular septal defect could be noted.

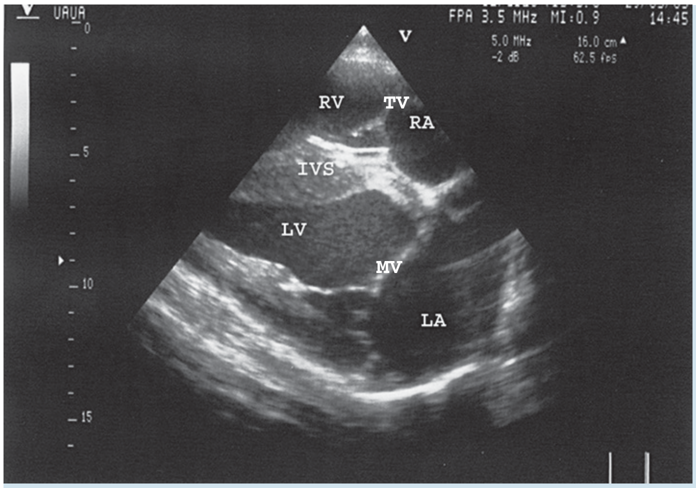


Figure 7.10: Ultrasonographic image of the right parasternal long axis 4 chamber view. This image corresponds to scan plane C in Fig 7.7. Structures seen with this view include: right atrium (RA), right ventricle (RV), tricuspid valve (TV), interventricular septum (IVS), left atrium (LA), left ventricle (LV), mitral valve (MV) and the LV free wall.

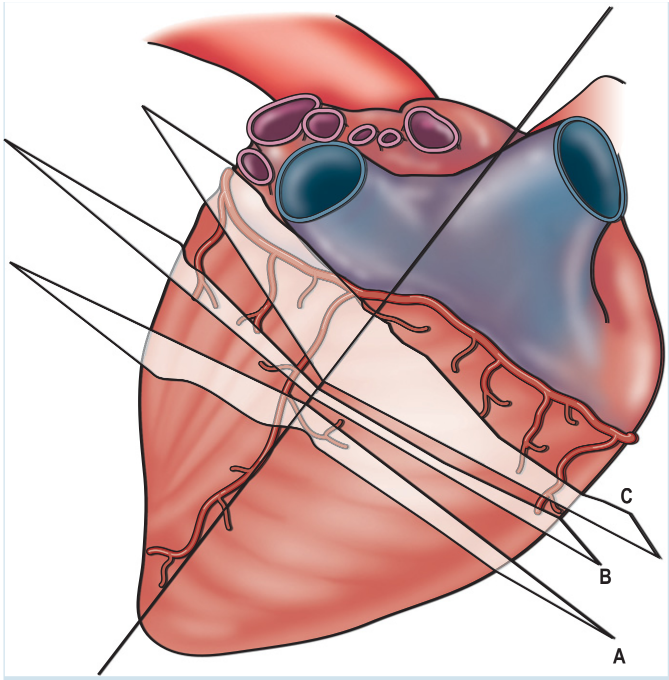


Figure 7.11: Scan plane of the short axis views obtained from the right cardiac window with the transducer positioned in the 4th intercostal space at the four o'clock position. (A) Scan plane of the short axis view of the left ventricle. The transducer is positioned in the 4th intercostal space at the four o'clock position and pointed ventro-caudally towards the 5th intercostal space. (B) Scan plane of the short axis view of the mitral valve. The transducer is positioned in the 4th intercostal space at the four o'clock position, perpendicular to the chest wall and pointed straight across the chest. (C) Scan plane of the short axis view of the aortic valve. The transducer is positioned in the 4th intercostal space at the four o'clock position and pointed upwards towards the withers.

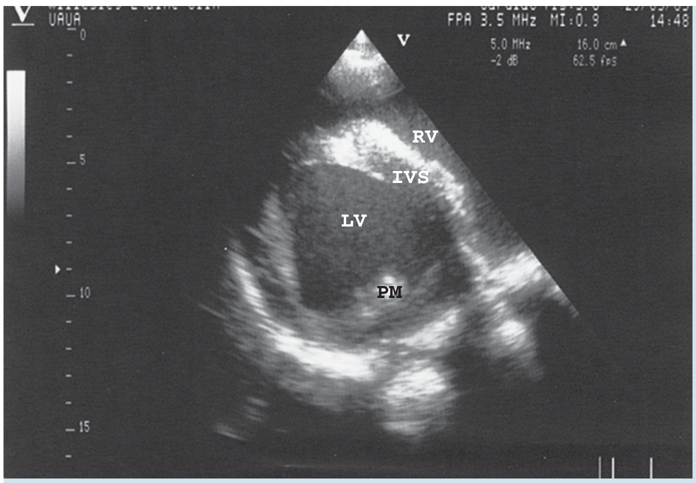


Figure 7.12: Ultrasonographic view of the right parasternal short axis view of the left ventricle. This image corresponds to scan plane A in Fig 7.11. Structures seen with this view include: left ventricle (LV), papillary muscles (PM), right ventricle (RV) and the interventricular septum (IVS).

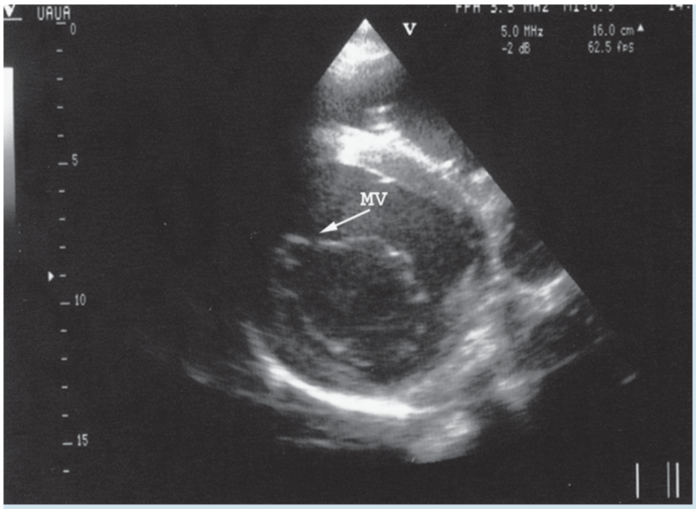


Figure 7.13: Ultrasonographic view of the right parasternal short axis view of the mitral valve. This is known as the "fish mouth view" which corresponds to the movement of the mitral values. This image corresponds to scan plane B in Fig 7.11. Structures seen with this view include: mitral leaflets (septal leaflet) and free wall leaflet as well as the right and left ventricles. This image shows the septal leaflet of the mitral valve (MV).

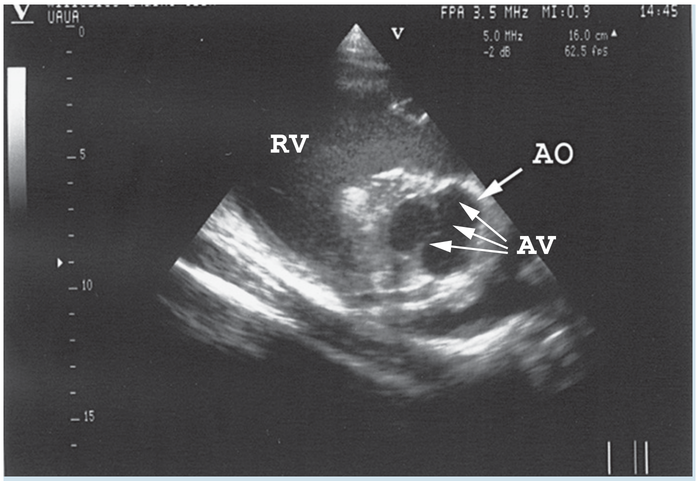


Figure 7.14: Ultrasonographic view of the right parasternal short axis view of the aortic valve. This view is also called the "Mercedes Benz view". This image corresponds to scan plane C in Fig 7.11. Structures seen with this view include the aorta (AO), the three cusps of the AV (right coronary cusp, left coronary cusp and the non-coronary cusp) and right ventricle (RV).

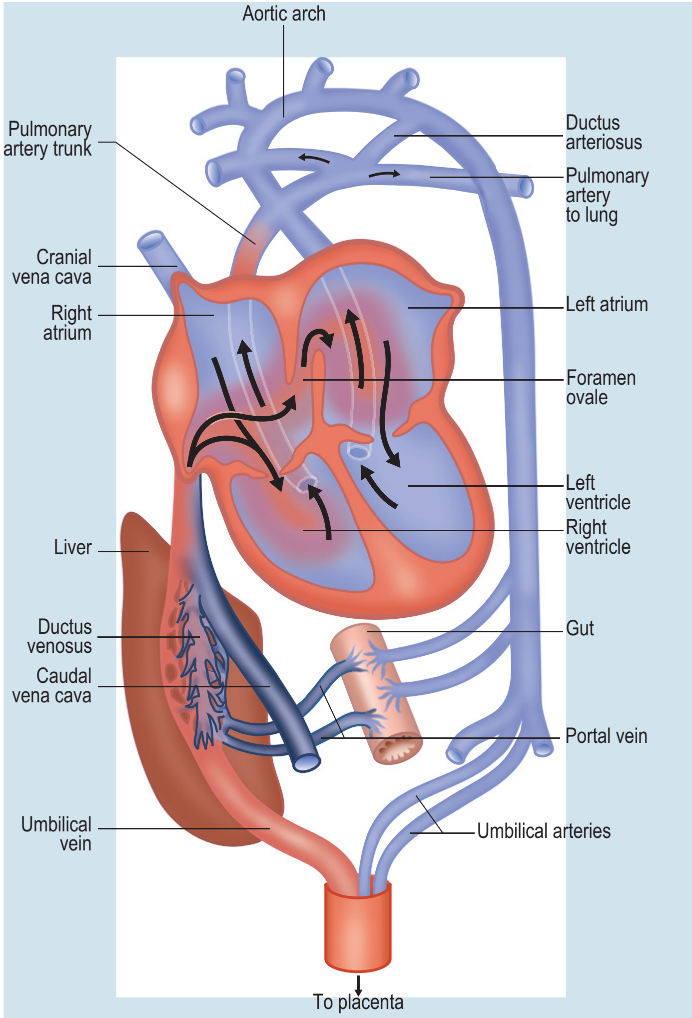


Figure 7.15: Schematic representation of fetal circulation. Oxygenated blood travels from the placenta in the umbilical vein (red). This oxygenated blood mixes with deoxygenated blood (dark blue) returning from the bowel and other organs in the caudal vena cava. Two thirds of this blood directly crosses the foramen ovale into the left atrium. Thus the best oxygenated blood available to the fetus is preferentially delivered to the heart and head. The remaining blood (a mixture of oxygenated and de-oxygenated – light blue) passes through the right ventricle into the pulmonary trunk and then through the ductus arteriosus to the descending aorta. During fetal life the right ventricle is dominant in both size and load and both ventricles work in "parallel" connected by the foramen ovale and ductus arteriosus.

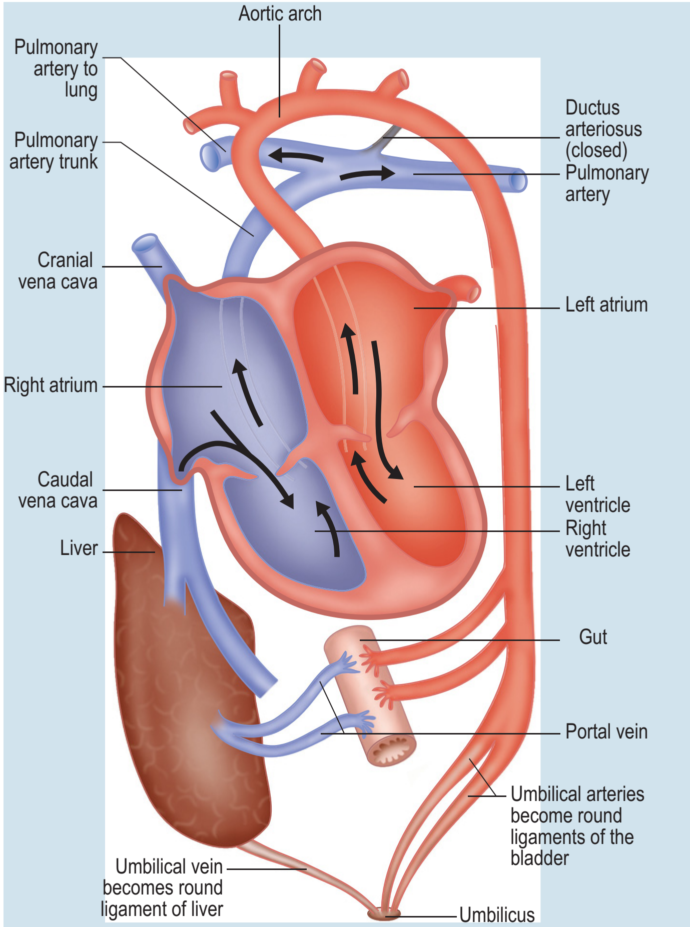


Figure 7.16: Schematic representation of post-natal circulation. At birth the lungs expand in response to increased partial pressure of carbon dioxide in the blood. This results in a decrease in pulmonary vascular resistance, which in turn reduces the pressure in the pulmonary artery compared to the aorta and thus blood is directed into the lungs. The increase in pressure in the left atrium results in closure of the foramen ovale. Closure of the ductus arteriosus after birth is mediated by several factors, including elevated oxygen tension and decreasing levels of circulating or tissue prostaglandins. However, due to its elastic nature, complete closure takes a number of days. If in that time hypoxemia develops the ductus arteriosus can re-open.

At the time the umbilical cord ruptures or is clamped/ligated, blood flow through the umbilical vein ceases, systemic vascular resistance increases and the smooth muscular sphincter controlling the blood flow through the ductus venosus constricts. These effectively close this fetal communication between the umbilical vein and inferior vena cava.

Congenital cardiac disorders

90% of newborn foals have been reported to have audible murmurs and 96% to have various arrhythmias including atrial tachycardia, ventricular premature contractures, A-V block, etc. which disappear by 15 minutes postpartum. Hypoxemia and elevated vagal tone likely account for many of these electrical disturbances.

Foals with widely radiating grade (III/V or greater) murmurs, cyanosis, jugular pulses, peripheral edema, ascites, pleural effusion or syncope, weakness, growth retardation and/or lethargy are suggestive of cardiovascular insufficiency.

Diagnoses typically are determined via echocardiography and/or contrast studies, blood gas analyses, thoracic radiography, and physical exam.

Persistent neonatal pulmonary hypertension (Fig 7.17)

If the "normal" anatomical and physiological transitions from fetal to neonatal circulatory orientation do not transpire, a condition of

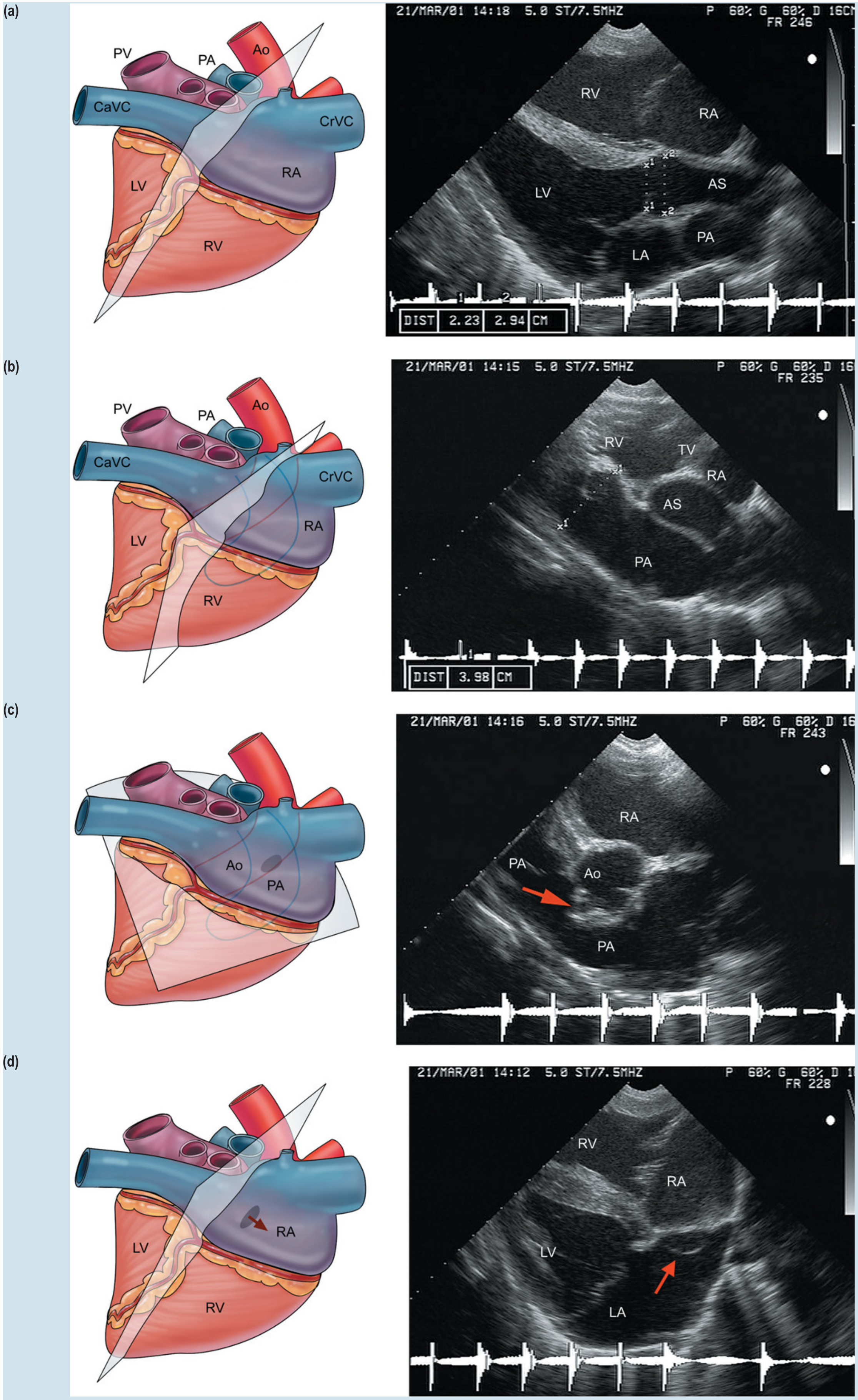


Figure 7.17: Right parasternal long-axis echocardiograms from a 6-hour-old Thoroughbred colt with persistent pulmonary hypertension. (a) This echocardiogram demonstrates the left ventricular outflow tract and a normal diameter of aortic valve and aortic sinus. (b) This echocardiogram demonstrates a dilated pulmonary artery. (c) This echocardiogram demonstrates a persistent ductus arteriosus (arrow). (d) This echocardiogram demonstrates a patent foramen ovale (arrow). AS = aortic sinus, Ao = aorta, CaVC = caudal vena cava, CrVC = cranial vena cava, PA = pulmonary artery, TV = tricuspid valve, LV = left ventricle, RV = right ventricle, LA = left atrium, RA = right atrium, PE = pericardial effusion. (From Corley KTT 2003 Monitoring and treating the cardiovascular system in neonatal foals. Clinical Techniques in Equine Practice 2:42–55.)

persistent pulmonary hypertension can arise. This has also been known as "persistent fetal circulation," "neonatal pulmonary vasospasm," "persistent transitional circulation," "persistent neonatal pulmonary vascular obstruction" or "neonatal pulmonary ischemia."

This condition results in systemic arterial hypoxemia due to increased pulmonary vascular resistance and shunting of pulmonary blood to the systemic circulation. Failure of the pulmonary vascular resistance to lessen postpartum despite alveolar expansion and alveolar oxygenation, encourages continued variable shunting of blood through the ductus arteriosus and/or foramen ovale with resultant systemic arterial hypoxemia. Factors that have been thought to predispose to persistent pulmonary hypertension in humans include:

- steroidal/non-steroidal use in the pregnant dam promoting repeated in utero closure of the ductus arteriosus
- pulmonary vasculature vasospasm as a result of hypoxia
- chronic placental insufficiency leading to intrauterine hypoxia and subsequent fetal pulmonary arteriolar hypertrophy
- regional alveolar hypoxia (i.e. meconium aspiration)
- increased tumor necrosis factor, platelet activating factor, leukotrienes, etc. causing active pulmonary vasoconstriction (i.e. sepsis)
- microthrombi (platelet-fibrin microthrombi, etc.) inducing pulmonary hypertension
- hypoglycemia
- hypocalcemia.

Diagnosis

If this condition is suspected the foal should be sent to a referral institute as diagnosis and treatments are difficult in the field.

- Following O_2 administration, a right foreleg (preductal) PaO_2 will likely show a tension difference/gradient (>20 mmHg) when compared to a hindlimb (postductal) if PDA shunting is substantial – this gradient may not be present however, if foramen ovale shunting is marked
- Thoracic radiography
- Cardiac echocardiography.

Treatment

- Increase alveolar O_2 ± mechanical ventilation to create a hypocapnic alkalosis $PaCO_2$ 25–35 mmHg, PaO_2 50–100 mmHg and pH 7.4–7.55.
- Decrease pulmonary hypertension/vasoconstriction:
  - inhaled nitric oxide
  - sildenafil 0.5–2.5 mg/kg PO q 24h.
- Maintain systemic blood pressure and perfusion through the use of intravenous fluids (crystalloids/colloids).
- Alkalinize – intracellular pH may be more important than $PaCO_2$ for alleviation of vasoconstriction.
- Vasodilation – examples include PGI_2 (prostacyclin) and tolazoline.
- Extracorporeal membrane oxygenation.

Patent ductus arteriosus (PDA) (Figs 7.18–7.21)

Otherwise normal foals may be noted to have a high pitched (machinery type), holosystolic, grade I–IV murmur associated with persistence of the ductus arteriosus (DA) that is best heard in the left 3rd–4th intercostal space. Ordinarily, this functional DA persistence is expected to close approximately 72–96 hours postpartum.

A PDA is typically a "left-to-right" shunt pushing aortic (high pressure) blood back through the DA into the pulmonary circulation. The aortic blood pushes through the patent ductus arteriosus, into the pulmonary artery to the pulmonary capillary beds, then returns to the heart via the pulmonary veins to enter the left atria and then ventricle where it is pushed back into the aorta and into the PDA again.

Consequently, a large persistent PDA typically results in enlargement of the left atrium and ventricle. Over time, if the pulmonary arterial pressure rises substantially, right-sided congestive heart insufficiency/failure may ensue.

Diagnosis

- Foals in which it is suspected that the PDA is functionally patent for greater than one week should have further cardiac diagnostic testing with echocardiographic and/or radiographic and blood gas evaluations.
- If supplemental oxygen is available, oxygen insufflation may be administered and simultaneous arterial blood gas comparisons between the right forelimb and a hindlimb made.
  - if the PDA enters the aorta distal to the brachiocephalic trunk, a differential oxygen gradient (PaO_2) of 20 mmHg or greater should be present between the right forelimb and a hindlimb
  - if the PDA enters the aorta more proximally or the foramen ovale is also persistently patent, this oxygen gradient difference may not exist.
- Clinically, foals with persistent large bore PDAs may have increased peripheral pulse pressure. This increase in pulse pressure is often described as "bounding" due to the left ventricular increased stroke volume and/or related systolic hypertension and decreased diastolic pressure from the aortic blood pushing into the pulmonary circulation via the PDA.

Treatment

Since the patency of the PDA is maintained by production of PGE_2 by the ductus arteriosus, prostaglandin suppression through cyclo-oxygenase inhibitors (i.e. flunixin meglumine, ibuprofen, indomethacin, etc.) may elicit closure of the PDA in some cases.

Ventricular septal defect (VSD) (Figs 7.22–7.28)

History and clinical signs

Reportedly VSD is the most frequently occurring congenital cardiac defect in large animals. *Foals with VSDs often have readily auscultable*

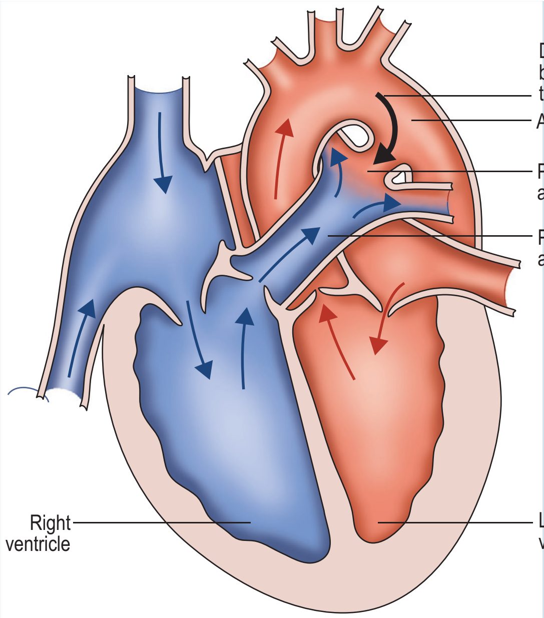


Figure 7.18: Schematic representation of a PDA.

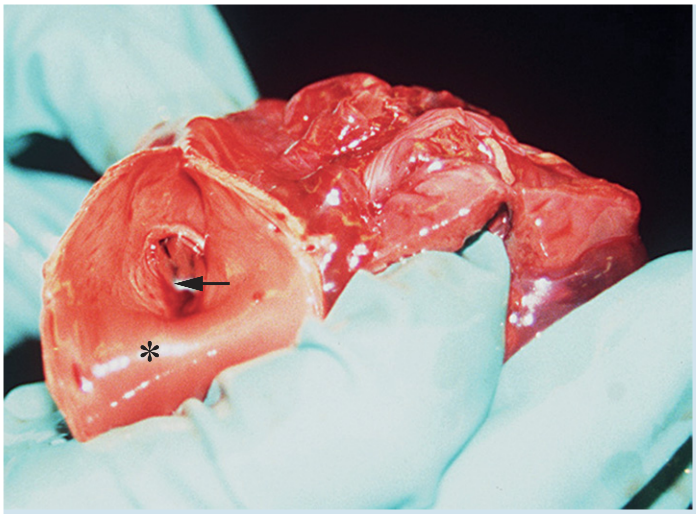


Figure 7.21: Postmortem specimen from a 6-day-old foal demonstrating a PDA (arrow). Note the aorta (*).

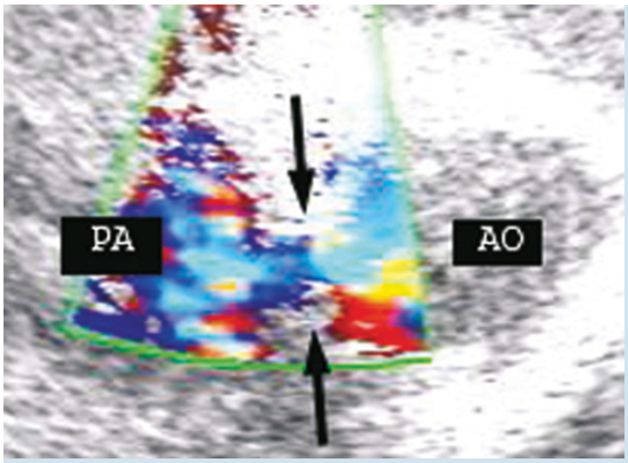


Figure 7.19: Color flow Doppler of a PDA in a 24-hour-old foal. An intracardiac shunt from the aorta to pulmonary artery is visible (arrows).

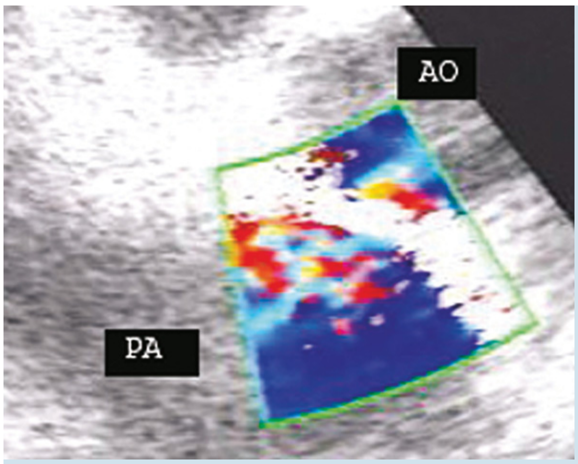


Figure 7.20: Color flow Doppler from the same foal 2 days later demonstrating resolution of the PDA.

murmurs on both sides of the thorax. A marked right-sided, radiating, Grade 5–6 systolic murmur best heard over the 4th intercostal space is typical. A less intense, left-sided systolic heart base murmur is often present.

Pulmonary valve outflow volume is increased due to the left-to-right shunting through the VSD and this increase in volume being ejected through the pulmonary aperture is responsible for the left systolic murmur often heard. A thrill is frequently palpable with sizeable VSDs.

The large majority are situated in the membranous portion of the interventricular septum just below the left heart's right coronary cusp of the aortic valve and the right heart's tricuspid valve.

Membranous defects that are smaller than 25 mm and demonstrate velocity of greater than 4 m/sec may be exercise tolerant.

Muscular ventricular septal defects, defects with aortic insufficiency, or other anomalies are likely to be intolerant of strenuous exercise.

Foals with membranous VSD larger than 35 mm in diameter or muscular VSDs tend to have decreased life expectancy.

VSD may also be a component of complex cardiac anomalies carrying a poor prognosis.

Treatment

Currently there are no treatment options available.

Diagnosis

- Peripheral signs of circulatory status and electrocardiographs are usually normal.
- Ultrasound studies including Doppler ± bubble contrast are the best diagnostic tools.
- Radiography can occasionally be used to demonstrate cardiac enlargement in cases with large defects.

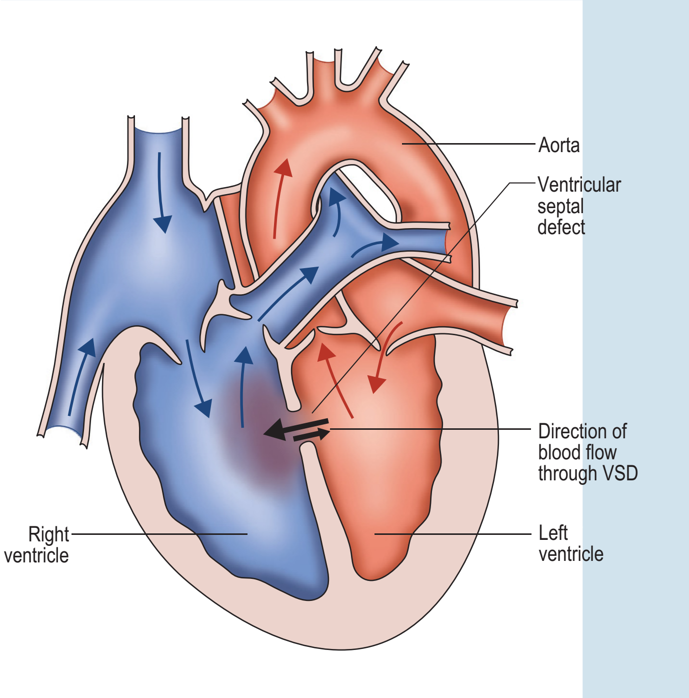


Figure 7.22: Schematic representation of a ventricular septal defect (VSD).

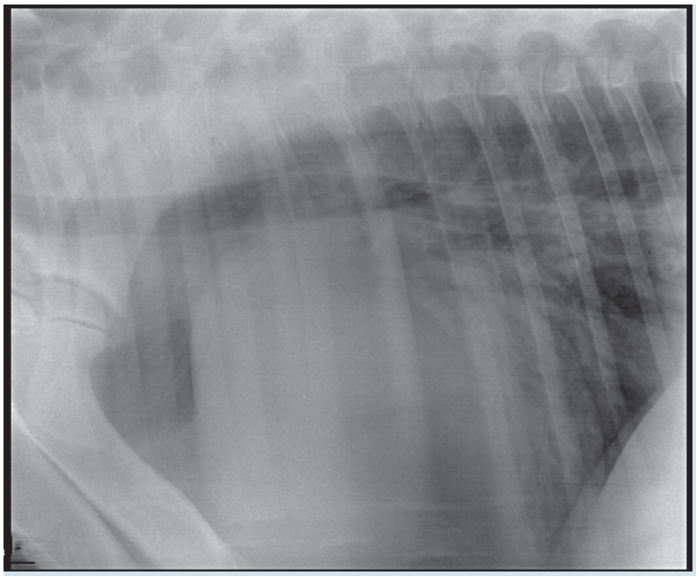


Figure 7.23: A lateral thoracic radiograph from a 6-week-old Thoroughbred foal with marked cardiomegaly due to a large ventricular septal defect. Note there is elevation of the trachea and the cardiac silhouette occupies the majority of the chest.

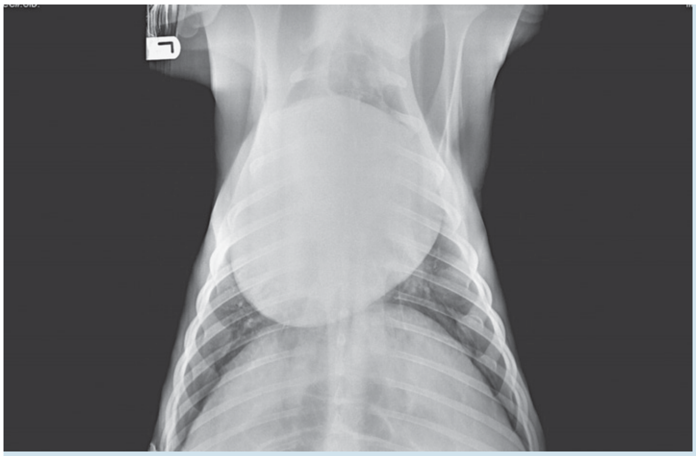


Figure 7.24: A dorsoventral thoracic radiograph from a 6-week-old Thoroughbred foal with marked cardiomegaly due to a large ventricular septal defect (same foal as Fig 7.23). The cardiac silhouette again occupies the majority of the chest.

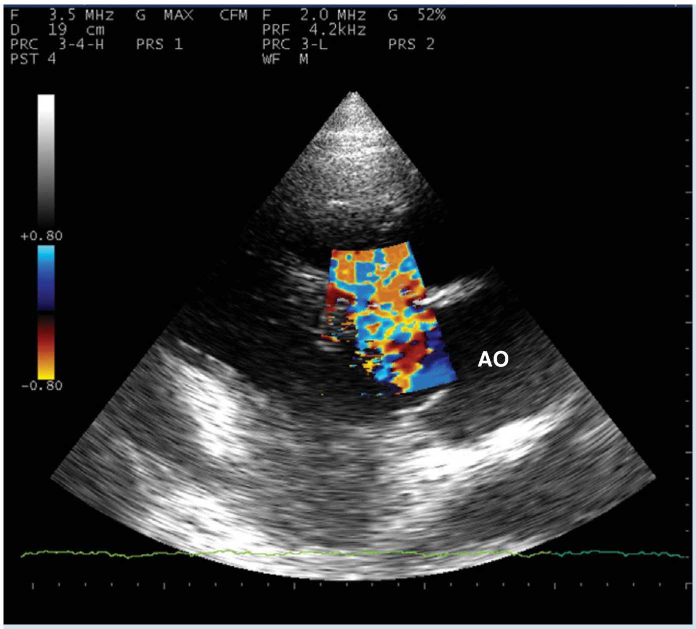


Figure 7.25: 4-month-old Standardbred colt that was noted to have a grade 4/6 right-sided systolic murmur and a grade 2/6 left-sided systolic murmur. The foal was noted to be healthy and of normal stature. Color flow Doppler examination revealed a ventricular septal defect in a sub-aortic location. Aorta (AO).

Atrial septal defect (ASD) (Fig 7.29)

Atrial septal defects are uncommon in the equine neonate. Most are a component of complex congenital cardiac anomalies.

Three types of ASD are most described – ostium primum, ostium secundum and sinus venosus. However, a fourth, "unroofed coronary sinus" which is a coronary sinus septal defect should also be mentioned.

- Atrial septal defects can occur low in the septum just above the ventricular septum (ostium primum defects). Defects in this area may involve a leaflet of the mitral valve as well.
- Defects that occur in the middle of the atrial septum (ostium secundum) are in the region of the foramen ovale.
- Defects in the upper atrial septum (sinus venosus) may involve one of the pulmonary veins. Specifically, a right pulmonary vein may empty into the right atrium instead of the normal entry point in the left atrium, creating an "anomalous pulmonary vein."
- Unroofed coronary sinus defects allow communication of venous coronary sinus blood with the left atrium due to an undeveloped septum between these two structures.

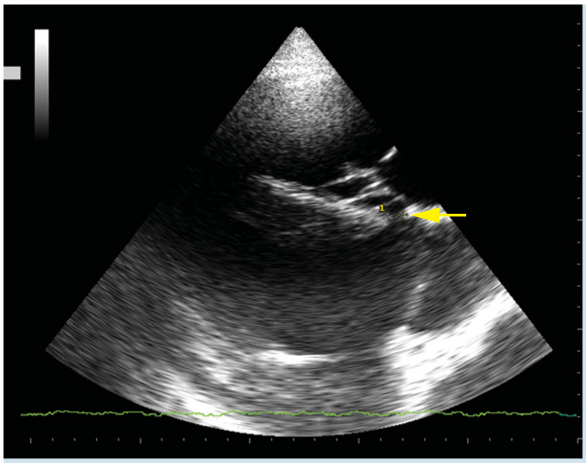


Figure 7.26: Same foal as Figure 7.25. The VSD measured <1 cm and the prognosis for athletic function was good. It was recommended that another echocardiogram be performed prior to entering training.

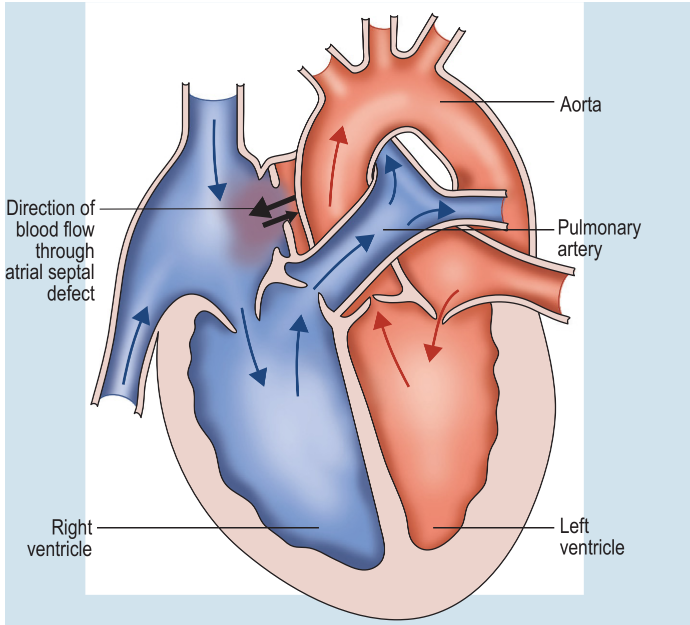
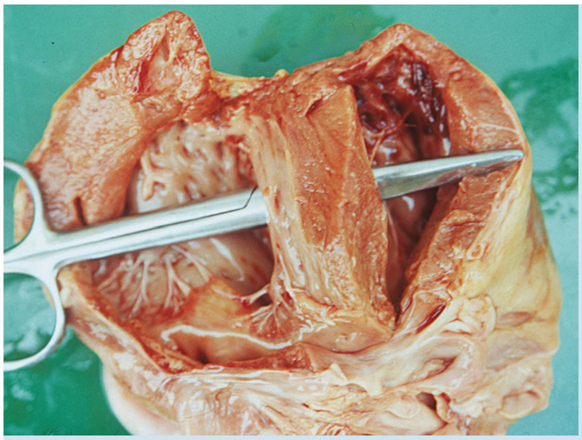


Figure 7.29: Schematic representation of an atrial septal defect (ASD).



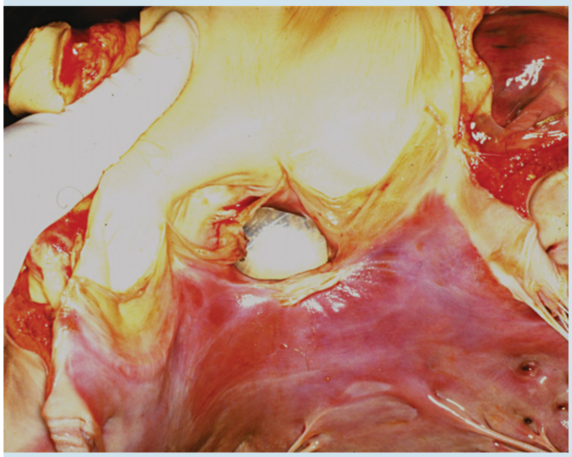


Figure 7.27 & 7.28: Postmortem specimens of a muscular VSD (above) and membranous VSD (below).

Diagnosis

- Foals often have a pronounced heart base murmur and are frequently exercise intolerant at presentation.
- Clinical signs of right-sided volume overload predominate (jugular pulsation, ascites, pleural effusion, peripheral venous distension and edema) and supraventricular arrhythmias are not uncommon due to right atrial enlargement.
- Definitive diagnosis is made with echocardiography. Color flow Doppler may be required to differentiate a residual, non-functional foramen ovale from a true ASD.

Tetralogy of Fallot (Figs 7.30–7.32)

Pulmonary valve stenosis, ventricular septal defect, overriding aorta, and right ventricular hypertrophy make up this complex cardiac defect. If a patent ductus arteriosus also is present, then pentalogy of Fallot is present. Right-to-left shunting results in cyanosis and lethargy.

Clinical signs and diagnosis

- Exercise intolerance, difficulty nursing and dyspnea.
- A loud grade 4/6 to 6/6 pansystolic murmur with palpable thrill over the left heart base with point of maximal intensity (PMI) over the pulmonary valve area.

Treatment

None – the prognosis is grave.

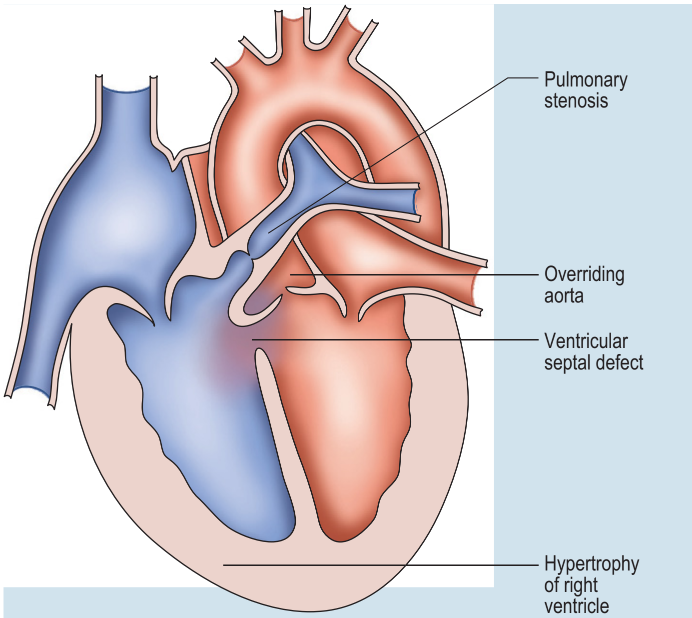


Figure 7.30: Schematic representation of tetralogy of Fallot.

Tricuspid atresia (Figs 7.33 & 7.34)

Tricuspid atresia is a rare defect in foals that often accompanies complex cardiac anomalies. Persistent foramen ovale, ventricular septal defect, ventricular dilation and mitral dysplasia have been reported with this defect.

Diagnosis

- Two-dimensional echocardiography demonstrates echo banding in the tricuspid valve region, fallout of the aortic root, enlarged left ventricle, small right ventricle, and variable atrial enlargement and mitral excursion variations.
- Contrast echocardiographic techniques confirm intracardiac shunting of blood from right to left atrial chambers then to left ventricle.

Great vessel transposition (Figs 7.35 & 7.36)

Failure of the trunco-conal septum to "spiral" downward during cardiac development leads to the aorta exiting the right ventricle and the pulmonary artery the left ventricle. Foals born with this anomaly may survive for several days postpartum but are markedly cyanotic and frequently recumbent.

Auscultation often reveals a louder right-sided systolic murmur compatible with VSD as well as the continuous crescendo–decrescendo "machinery" type left heart base murmur of the PDA.

In addition to the great vessels being "transposed," a ventricular septal defect, functional foramen ovale, and patent ductus arteriosus are often present and are responsible for the foal's brief survival. If shunting of blood does not occur through these structures, this anomaly is incompatible with life.

Prostaglandin inhibition via non-steroidal anti-inflammatory medications and subsequent pharmacological closure of the patent ductus arteriosus can be fatal for these foals if this life preserving extracardiac shunt closes.

Diagnosis

Echocardiography demonstrates a characteristic "twin pipe" appearance as the two outflow tracts exit the ventricles in near parallel instead of their normal twisted configuration.

Treatment

No effective therapy is available for equids and currently humane euthanasia is recommended.

Truncus arteriosus (Fig 7.37)

Incomplete or failed septation of the embryonic truncus arteriosus leads to a common "trunk" through which the aorta and pulmonary artery flow. A ventricular septal defect is also evident. This leads to mixing of oxygenated and non-oxygenated blood, variable cyanosis and often pulmonary vascular overload. Decreased PaO_2, right heart insufficiency and pulmonary edema often ensue. Since pulmonary vascular resistance is lower than systemic vascular tone, blood is preferentially pushed into the pulmonary vascular system, driving pulmonary vascular overload with resultant clinical signs.

Clinical signs

- Exercise intolerance
- Stunted growth
- Dyspnea or syncope may be noted
- Grade 4/6 to 6/6 crescendo–decrescendo cardiac murmur with palpable thrill over left heart base.

Cardiac ectopia

Rare congenital condition in which the heart is situated partially or entirely outside the thoracic cavity. Four classifications are described – thoracic, thoracoabdominal, abdominal and cervical. Most foals such affected are aborted and this anomaly is often not compatible with normal fetal development. Frequently, this defect is associated with concurrent defects such as atrial and septal ventricular defects, tetralogy of Fallot, valvular atresias, valvular stenoses, and pericardial anomalies.

Coagulation defects (Figs 7.38–7.40)

Several congenital clotting defects in horses have been reported. However, all of these are very rare. They include:

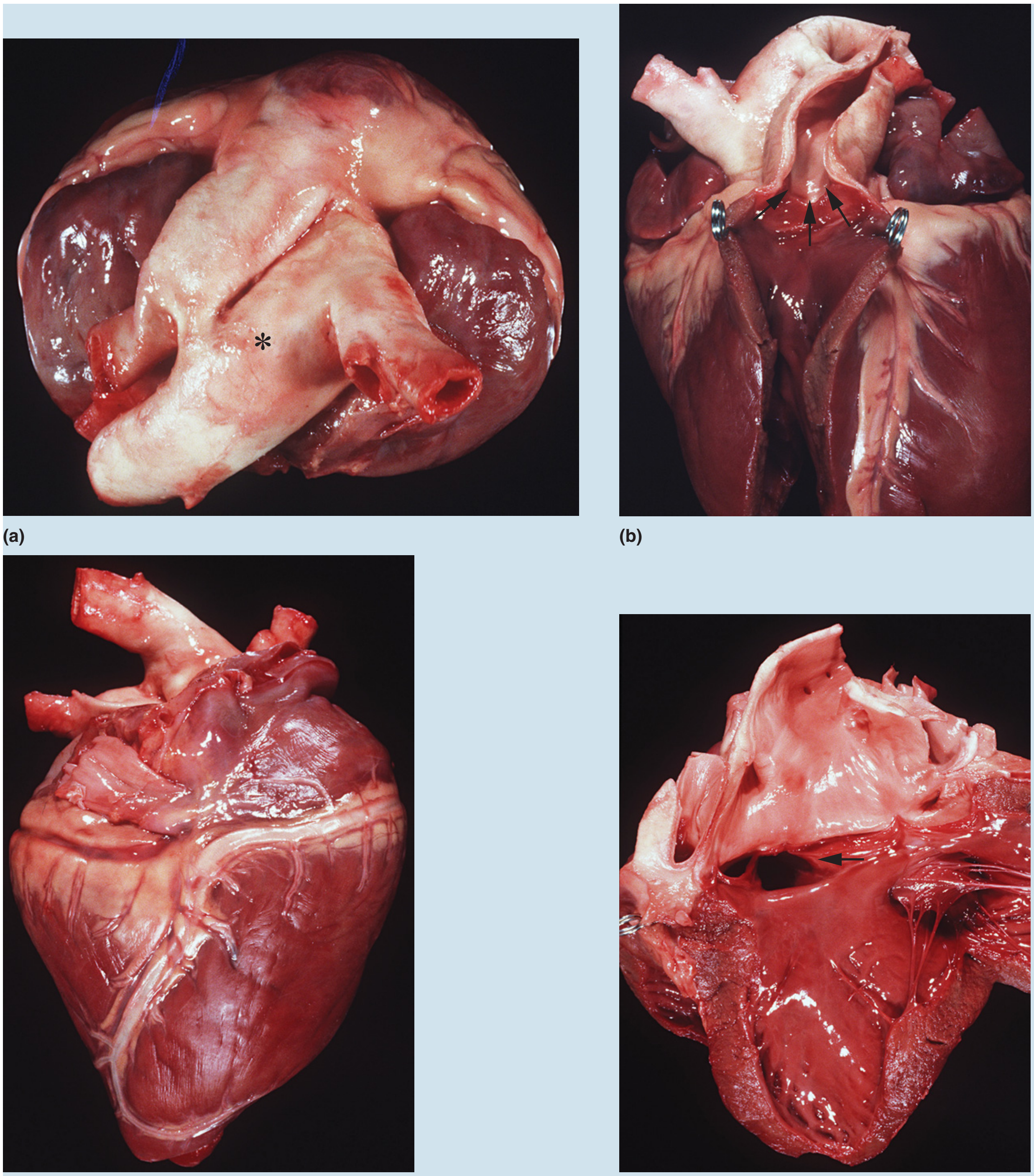


Figure 7.31: Postmortem specimen demonstrating a tetralogy of Fallot. (a) Note dextroposition of aorta (*). (b) Note pulmonary stenosis (arrows). (c) Note right ventricle hypertrophy (right side of image). (d) Note ventricular septal defect (arrow).

- Protein C deficiency – reported in one Thoroughbred. Protein C is a vitamin K-dependent molecule which is a natural hemostatic inhibitor.
- Prekallikrein deficiency – has been reported in Belgians and miniatures. Prekallikrein is needed for the activation of the intrinsic coagulation cascade. It is one of several contact activation factors that interacts with the negative charged surfaces (e.g. collagen) in the subendothelial matrix.
- Von Willebrand's disease – has been reported in Thoroughbreds and Quarter horses. Laboratory clotting times are normal. This disease results in failure of platelet aggregation and activation resulting in increased tendency to bleed.
- Hemophilia A (inherited factor VIII deficiency):
  - most common clotting deficiency encountered
  - sex-linked – only males are affected but females are carriers.
  - has been reported in a number of breeds.

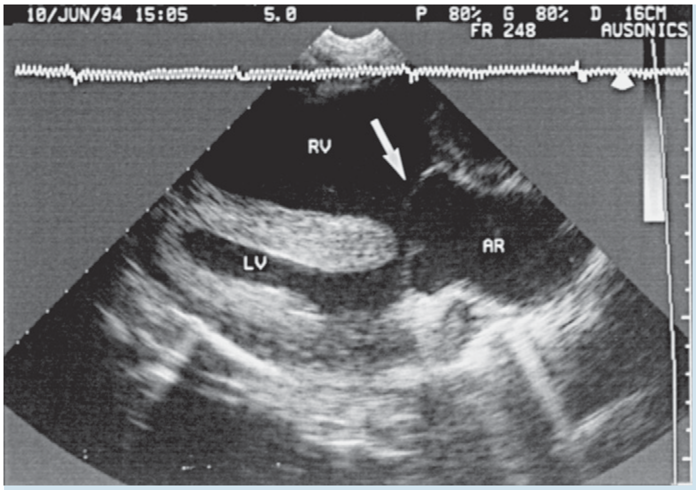


Figure 7.32: Echocardiogram from a 1-week-old Arabian-Thoroughbred-cross filly with tetralogy of Fallot. Long axis view of the left ventricular outflow tract demonstrates the large ventricular septal defect (arrow), the overriding aorta (AR), and right ventricular dilatation and hypertrophy. RV, right ventricle; LV, left ventricle.

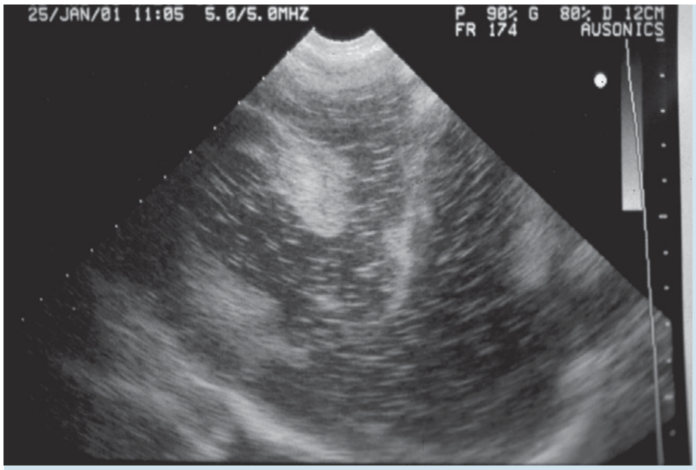


Figure 7.34: Same foal as Figure 7.33. Bubble contrast study demonstrating the admixture of venous and arterial blood. The foal was euthanized due to a hopeless prognosis.

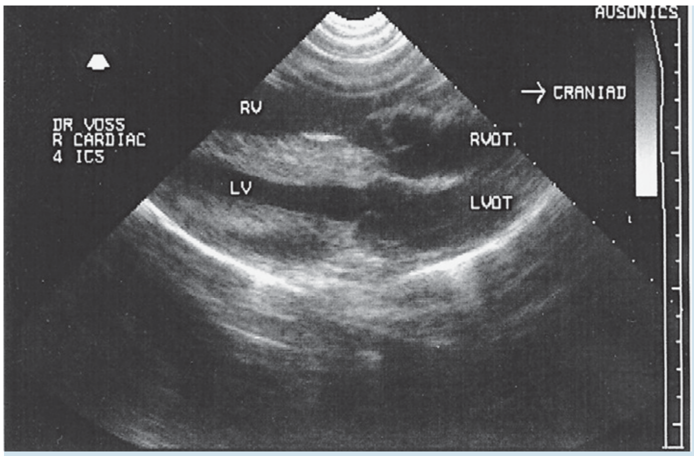


Figure 7.36: Great vessel transposition – this is an echocardiographic from a 3-day-old Arabian colt with cyanosis, exercise intolerance and lethargy. Note the "twin pipe" parallel arrangement of the transposed aorta and pulmonary artery. A small membranous VSD, patent ductus arteriosus, and open foramen ovale were also present.

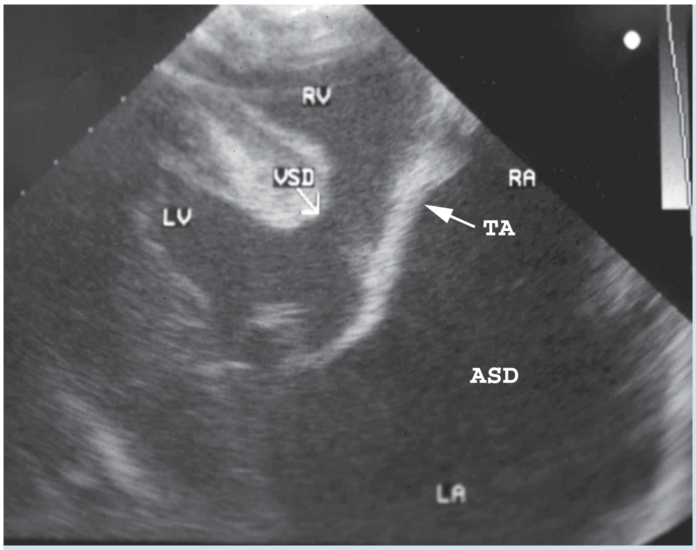


Figure 7.33: Tricuspid atresia (TA), atrial septal defect (ASD), ventricular septal defect (VSD), and mitral valve dysplasia. This 3-hour-old foal presented with history of cyanosis despite being placed on appropriate nasal oxygen. No significant murmurs were noted with this foal's defect. The reason that no murmurs could be auscultated was that the pressures were equal in all chamber compartments.

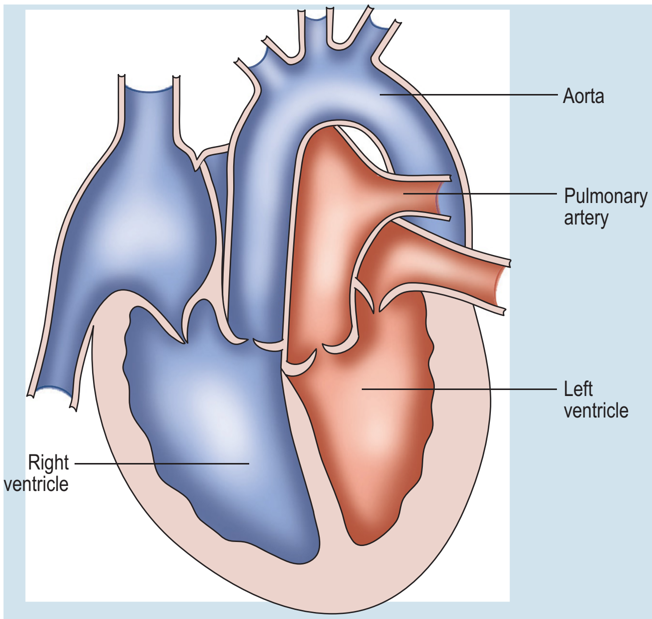


Figure 7.35: Great vessel transposition.

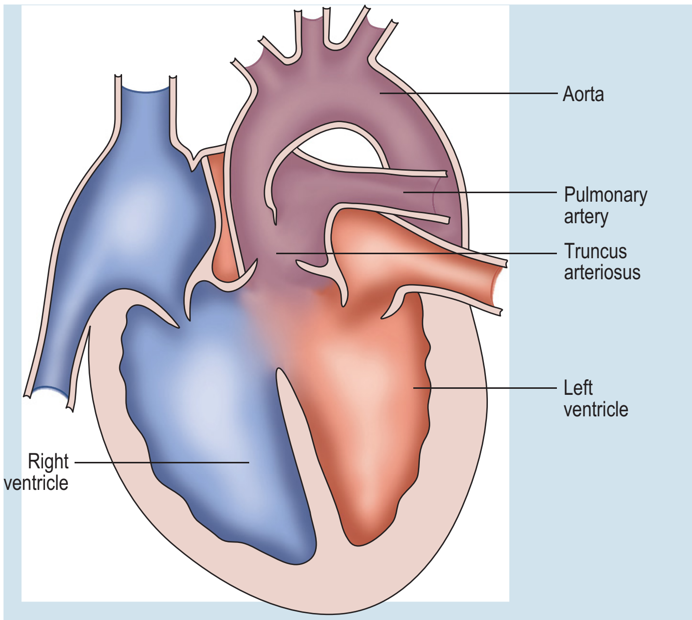


Figure 7.37: Truncus arteriosus.

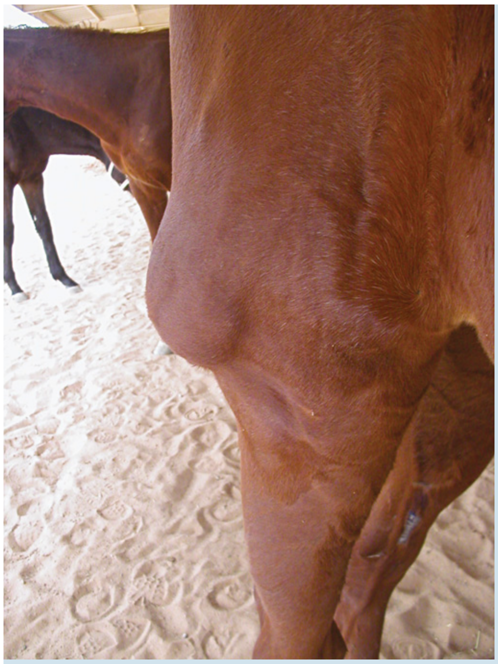


Figure 7.38: Hematoma above the stifle.

Figure 7.39: Hematoma at the point of the shoulder.

Figure 7.40: Spontaneous epistaxis.

Clinical signs

- Prolonged bleeding after minor trauma or routine procedures, e.g. passage of a nasogastric tube can result in severe and prolonged hemorrhage
- Hemorrhages into body cavities and joints as well as multiple hematomas can occur spontaneously or as a result of minor trauma
- Other signs include petechial hemorrhages, melena, anemia or frank blood in the urine

Diagnosis

A series of laboratory tests can be used to establish a diagnosis of a clotting disorder and to distinguish between the various disorders:

- Bleeding time (BT) – the time taken for a small cutaneous or gingival pinprick to stop bleeding. Normal time is 3–4 minutes.
- First stage prothrombin time (PT).
- Activated partial thromboplastin time (APPT).
- Clotting time (CT) – this is an in vitro test and is the time taken for a blood sample to clot in a glass tube at 37°C. Normal values are between 4.5 and 7 minutes.
- Factor VIII determination.

Von Willebrand's disease is diagnosed on the basis of measurement of plasma concentrations of von Willebrand factor (vWF) : Antigen.

Table 7.1 outlines the expected test results with each disorder.

Table 7.1: Diagnostic features of the more common congenital clotting disorders

	DEFICIT	BT (min)	CT (min)	PT (s)	APTT (s)
Hemophilia A	Factor VIII	>20	Prolonged	Prolonged	Normal
Von Willebrand's disease	vWF	Normal	Normal	Normal	Normal
Reference		2–4	4–7	9–12	47–73

Treatment and prognosis

- Emergency treatment is often undertaken in a hemorrhaging foal prior to diagnosis. Such treatments can include plasma or whole blood administration. However, catheter placement in these foals can in itself result in severe hemorrhage.
- There is no effective long-term treatment for these disorders and euthanasia is generally recommended.

Acquired cardiac disease

Arrhythmias

Following physical examination and careful auscultation of the heart, an electrocardiogram should be performed if an arrhythmia is suspected

Generally, a base apex lead system can easily and readily be applied to obtain a rhythm trace. To apply base apex leads (3 leads), the left arm lead is placed either behind the left elbow or near ventral midline behind the left elbow. The right arm lead is placed in the bottom of the right jugular furrow or right lateral neck. The left leg lead is placed on either side of the neck as the ground lead. An ECG machine set to record lead I or lead III will have the largest wave forms with this set up and should yield a good rhythm strip (see Fig 7.6).

Sinus arrhythmia

Two types of sinus arrhythmia are described:

- *Respiratory* – in which the heart rate increase during inhalation and decreases during exhalation. Variability of R–R intervals occurs with respiratory pattern, slowing the heart with expiration and accelerating the heart with inspiration. Horses typically do not show a respiratory driven sinus arrhythmia but may demonstrate non-respiratory type sinus arrhythmias.
- *Non-respiratory* – occurs independent of the respiratory cycle such that R–R interval variations are random. Non-respiratory sinus arrhythmias are not uncommon findings with diseases causing vasospasm, hypoxemia, electrolyte disturbances, or increased intracranial pressure. Wandering pacemaker and premature atrial beats are also sinus arrhythmias within this category. This group of arrhythmias is often asymptomatic and not treated directly in the neonatal foal. Therapy of identifiable underlying systemic disease is advised with anticipated resolution of these arrhythmias as the foal ages. In one study, approximately 60% of neonatal thoroughbred foals displayed sinus arrhythmias (32/50 wandering pacemaker, 30/50 premature atrial beats) that were attributable to hypoxemia and elevated vagal tone.

Sinus tachycardia

Elevated heart rate (>100 beats per minute) as a result of rapid sinus node impulses is sinus tachycardia. Usually this is a result of increased catecholamine release (or exercise) and is a physiologic response.

Pyrexia, hypoxemia, hypotension/hypovolemia, sepsis, or stimulant class medications can trigger sinus tachycardia. Withdrawal of adrenergic drugs and treatment of identifiable systemic abnormalities should resolve this arrhythmia unless cardiac insufficiency is manifest.

Sinoatrial arrest (Fig 7.41)

Sinus arrest is an uncommon disorder. On auscultation it sounds like a second degree AV block. Sinus arrest can be a parasympathetic manifestation [high vagal tone blocking the pacemaker cells of the sinoatrial node (SAN)] in the horse but can also herald SA nodal disease. The atria do not contract and the ECG shows a period during which neither P waves nor QS/T complexes are generated.

- The block should dissipate with exercise.
- Often, marked bradycardia, and/or frequent arrest periods are associated with pathology within the SA node.
- Vagolytic medications and exercise would be expected to ameliorate parasympathetic drive and arrhythmia if vagally mediated.

Atrioventricular block (Fig 7.42)

Disruption or delay of electrical impulses from the atria to the ventricles is termed A-V block. Three degrees of A-V block are described.

- In first degree A-V block, the conduction is only delayed but conducted. It is a physiologic lengthening of the P–R interval and

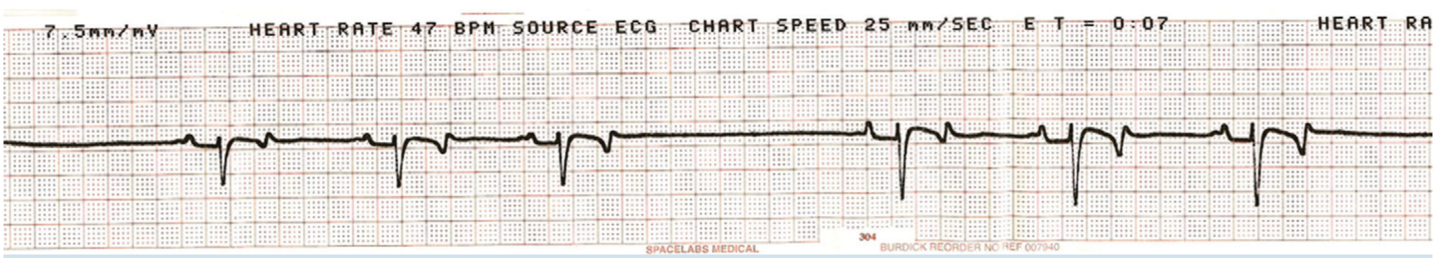


Figure 7.41: Sinus block in a foal that was born after a prolonged dystocia; 20 minutes later the sinus block disappeared.

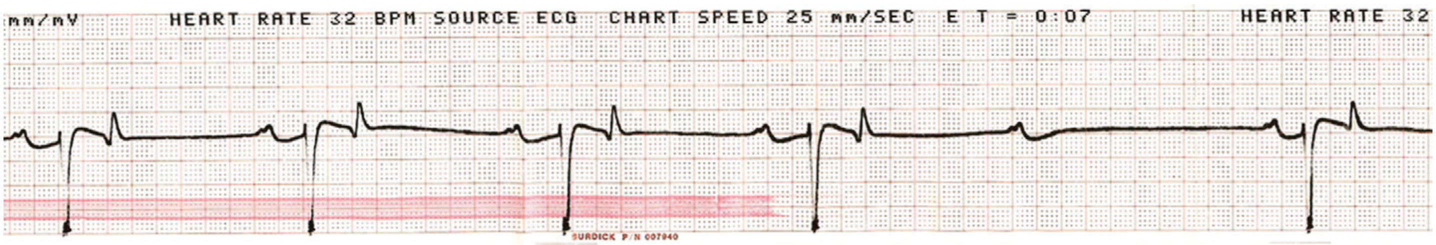


Figure 7.42: Mobitz type 1 second degree A-V block. Note the prolongation of the PR interval until the beat is dropped. After exercise the arrhythmia could not be detected.

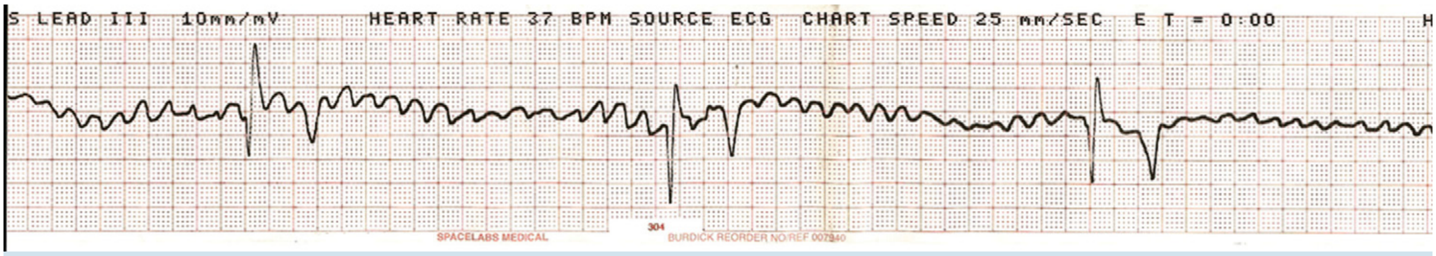


Figure 7.43: Atrial fibrillation in a 3-month-old foal with severe diarrhea and hyponatremia. The atrial fibrillation resolved once the animal's electrolytes were stabilized. Atrial fibrillation is caused by inhomogeneity of depolarization of the atrial myocytes. Coordinated atrial contraction does not occur and thus the sound of S4 is never heard during atrial fibrillation. This is a classic ECG revealing no definitive P waves, F waves (flutter) of the atria in the baseline and normal appearing QRS complexes at irregular intervals.

is resolved with exercise. Hence the P–R interval is prolonged (>0.5 sec) due to delayed conduction through the A-V node.

- Second degree block can be within the A-V node (Type I Mobitz) or the origin of the His bundle (Type II Mobitz) but both prevent the atrial depolarization from reaching the ventricular conduction system resulting in a P wave with no corresponding QRS.
  - Mobitz Type 1 – the P–R interval lengthens before the P wave is blocked, resulting in an absent QRS complex. This is a result of increased vagal tone and is considered a benign arrhythmia which disappears with exercise or excitement.
  - Mobitz Type 2 – the P–R interval is the same before and after the blocked P waves. It is considered a pathological arrhythmia.
- Third degree A-V block results in atrial and ventricular disassociation such that no electrical atrial impulses are conducted to the normal ventricular conducting system.

Approximately 14% of neonatal thoroughbreds may exhibit A-V block which resolves within 15 minutes following the postpartum adaptive period.

Atrial fibrillation (Fig 7.43)

In one study 30% of immediate postpartum Thoroughbred foals exhibited transient atrial fibrillation. Return to normal rhythm spontaneously occurred within 15 minutes postpartum in these foals. Hypokalemia and other electrolyte abnormalities which are frequently seen in foals with diarrhea predispose the equid to atrial fibrillation – as can excessive resting vagal tone.

Diagnosis

- On auscultation it is classically described as an "irregularly irregular" rhythm
- Atrial fibrillation is easily identified on an ECG strip as multiple rapid atrial "f" waves preceding normal appearing QRS complexes at irregular intervals.

Treatment

- Treat any underlying electrolyte abnormalities.
- Therapy with β-blockers, class 1A anti-arrhythmics (quinidine, procainamide, etc.), class 1C (flecainide, etc.), and class III medications (amiodarone, etc) should be reserved for intractable or persistent atrial fibrillation (>48 hours' duration) without spontaneous conversion to normal rhythm or with clinical signs of exercise intolerance, lethargy, etc. (See Appendix 5.)
- Therapy, i.e. conversion to normal rhythm, should be considered for most cases of paroxysmal atrial fibrillation of greater than 2 weeks' duration.

Premature atrial beats (PAB) (Fig 7.44)

Premature atrial depolarizations arise within atrial foci not often associated with the SA node. Therefore, atrial P waves of often variable morphology occur earlier than expected in the ECG recording. Most have a normal QRS that follows. Infrequent premature atrial beats can be found in apparently normal horses whereas frequent or paroxysmal PABs often herald supraventricular disease (organic or inflammatory in nature).

Therapy is not often recommended unless atrial tachycardia arises. Atrial tachycardia is defined as more than four consecutive premature atrial beats. Transient atrial tachycardia can occur in structurally normal hearts during exercise, catecholamine release, hypoxia, hypovolemia, electrolyte alterations and caffeine therapy due to increased automaticity or triggered activity. In one study 6% of Thoroughbred foals displayed transient atrial tachycardia which resolved spontaneously. Sustained atrial tachycardia, often of the re-entrant type, occurs with valvular/structural or congenital heart disease.

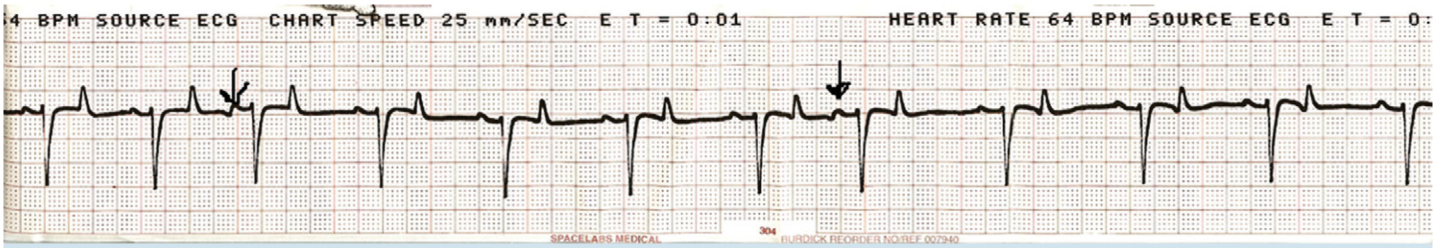


Figure 7.44: Atrial premature beats (arrows) in a 3-week-old foal. The foal had been bitten 3 days previously by a Western Diamond Back Rattlesnake. The foal was in DIC and had to be hospitalized for 2 weeks; 6 weeks after initial presentation the arrhythmia resolved.

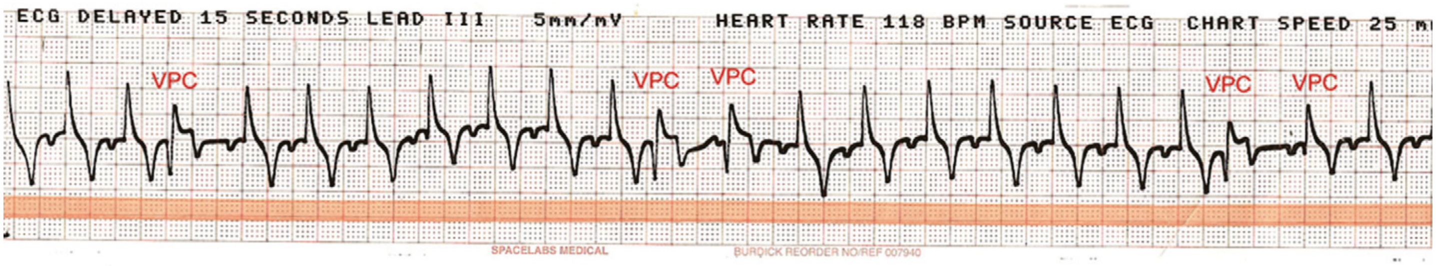


Figure 7.45: Ventricular premature contractions (VPC). Characteristic "bizarre" QRS complexes with lack of a preceding "P" wave. The QRS complexes are considered monomorphic and are thus assumed to arise from an isolated focus within the ventricles. This ECG was performed on a foal immediately post dystocia. Eight hours after appropriate fluid therapy and placement on nasal oxygen the tachycardia (120 BPM) and VPC had resolved.

Ventricular premature contractions (VPC) (Figs 7.45–7.47)

These premature contractions arise within the myocardium of the ventricles. Reportedly, premature ventricular contractions can transiently occur in approximately 20% of normal neonatal Thoroughbreds. Isolated or rare VPCs can be observed occasionally in normal individuals at rest or immediately post-exercise. Premature ventricular contractions that occur during exercise or are occurring frequently (>7 VPCs per minute) are abnormal. These VPCs can be caused by hypoxia, hypokalemia, hypomagnesemia, hypercalcemia, elevated catacholamines, adrenergic medications, endotoxemia/sepsis, and primary cardiac disease (myocardial, pericardial or contusive cardiac insults).

Clinical signs

- Early beats can be heard via auscultation and pulse deficits are common.
- A pronounced jugular pulse with the VPC may be observed due to atrioventricular asynchrony (cannon A wave) and the follow-up beat after a VPC may be stronger due to the increased left ventricular filling that occurs with a compensatory pause following the PVC. This is known as extrasystolic potentiation leading to greater intensity of this beat.
- ECG findings reveal lack of P waves preceding wide and/or tall ("bizarre") QRS complexes. If the QRS complex morphology varies, these PVCs are considered polymorphic and are assumed to arise from multiple/variable foci within the ventricles indicative of more diffuse myocardial disease.
- A physical examination, complete blood count, serum chemistry, blood gas (arterial ± venous), echocardiogram and ECG evaluations should be considered to define the inciting cause(s).

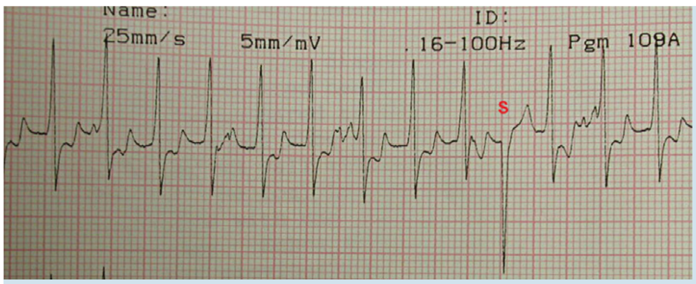


Figure 7.46: A 3-month-old Paint filly that presented with a 3-day history of lethargy, increased abdominal component with breathing and ventral edema. On arrival the filly had a heart rate of 140 BPM, marked crackles noted in all airways and excessive serous discharge from both nares. An echocardiogram revealed dilated cardiomyopathy with decreased fractional shortening of 12%. The ECG revealed ventricular tachycardia with a rare sinus beat (S).

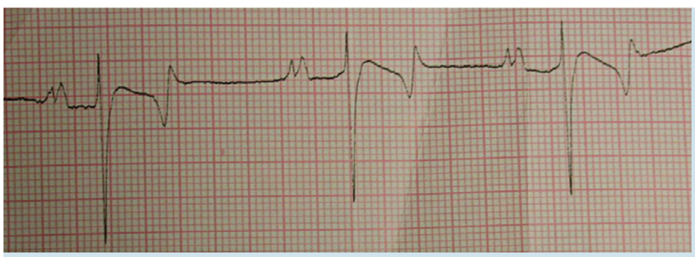


Figure 7.47: The foal in Fig 7.46 was converted with lidocaine as a slow infusion over 2 minutes for a total dose of 1.3 mg/kg IV. Sinus rhythm was achieved but fractional shortening had worsened after conversion (10%). This phenomenon has been documented in dogs after conversion and the foal should likely gain contractility over time. Monthly echocardiograms for 12 months revealed increased fractional shortening to 22%. The foal was of normal stature but the prognosis for an athlete was considered poor. The cause of this ventricular tachycardia was unknown.

- Evaluation of electrolyte abnormalities, as well as measurement of ionized calcium and magnesium, is recommended. Hypokalemia and hypomagnesemia are frequent causes of VPCs and electrolyte derangements may affect the usefulness of specific antidysrhythmic agents such as lidocaine.
- Measurement of cardiac troponin I and other cardiac enzymes can be used to document underlying cardiac disease. 95% of normal healthy foals in one study had cardiac troponin I values < 0.49 ng/mL. Values ≥ 1 ng/mL are highly suggestive of myocardial disease/inflammation.

Treatment

- Patients with abnormal VPCs should be closely monitored such that *ventricular tachycardia* (VT) does not arise i.e. heart rate >100–120 BPM with the rhythm being initiated from below the bundle of His (ventricular origin). Although potassium imbalance (both hypo- and hyperkalemia), other electrolyte disorders (hypomagnesemia), toxicants, vitamin E/selenium deficiency and stimulants can lead to VT, primary myocardial disease is most often associated with ventricular tachycardia.
- Therapy should be directed at the inciting cause with primary myocardial disease a likely complicating issue. Class I anti-arrhythmic medications (lidocaine, procainamide, quinidine, propafenone) are often effective in VPC patients with clinical signs of cardiac insufficiency while at rest. If multimorphic VPCs are noted, R waves are superimposed onto T waves on ECG or if the heart rate is excessive for the patients group therapy should be initiated.
- Although not as rapidly acting as lidocaine, magnesium sulfate (2–4 mg/kg q2 min) can be useful to control VPCs with a total dose not to exceed 50 mg/kg.
- The use of corticosteroids may be indicated if primary cardiac disease is deemed to be the inciting cause.

Ruptured chordae tendinae (Figs 7.48 & 7.49)

Traumatic rupture of the valvular chordae tendonae typically elicits pronounced mitral valve insufficiency with acute onset murmur and/or cardiac insufficiency (edema, atrial enlargement, arrhythmias) and in some cases collapse and death. This is more common in the author's experience in mid- to later-age horses but can occur sporadically in foals. Treatment is supportive and dictated by the cardiovascular state although the prognosis is frequently poor. Echocardiography is diagnostic.

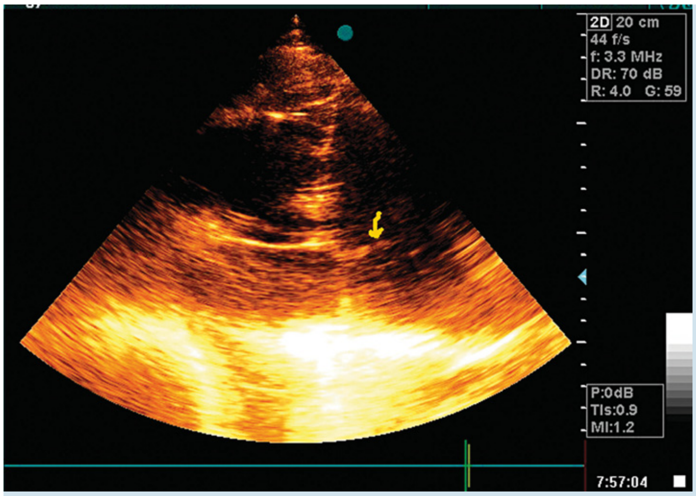


Figure 7.48: Ruptured chordae tendinae of the mitral valve in a 6-month-old foal. The foal has some changes on the mitral valve suggestive of endocarditis that probably led to the disruption of its chordae tendinae. Note the mitral valve prolapse – caudal accessory leaflet (arrow). The foal was euthanized because of his congestive heart failure.

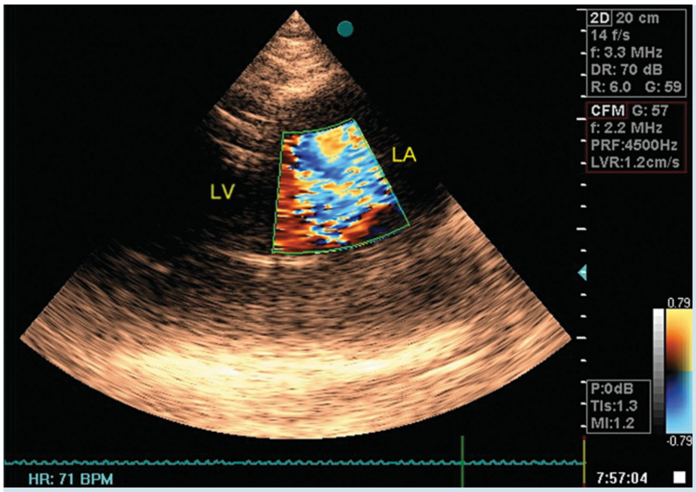


Figure 7.49: Color flow Doppler illustrating the jet lesion into the left atrium. On necropsy the endocardial surfaces of the left atrium are markedly thickened by fibrous connective tissue.

Endocarditis (Figs 7.50–7.57)

Bacterial endocarditis can affect the endocardium and have a mural distribution or be localized to one or more valve components. Microthrombi production during severe illness i.e. sepsis, etc. can lead to turbulent or traumatic endocardial damage which allows adhesion by transient bacteremic organisms and subsequent infective endocarditis. Thrombophlebitis and catheter sepsis have been associated with the development of endocarditis in the horse.

Clinical signs

- Most foals with bacterial endocarditis present with pyrexia, malaise and clinical indications of cardiac insufficiency attributable to the chamber/side of heart affected.
- Valvular lesions often incite regurgitant murmurs best heard over the valve involved. Therefore, atrioventricular valve endocarditis results in a systolic murmur whereas aortic or pulmonic valve endocarditis results in a diastolic murmur. Mural endocarditis and small vascular lesions may not produce heart murmurs, especially if the right side of the heart is affected.
- Right-sided vegetative lesions that dislodge may create embolic pneumonia or focal pulmonary abscesses whereas left heart vegetations can dislodge to spread septic emboli to other organ systems.

Diagnosis

Diagnostics include serial blood culture, CBC, chemistry, echocardiography, and thoracic radiography. Complete blood counts

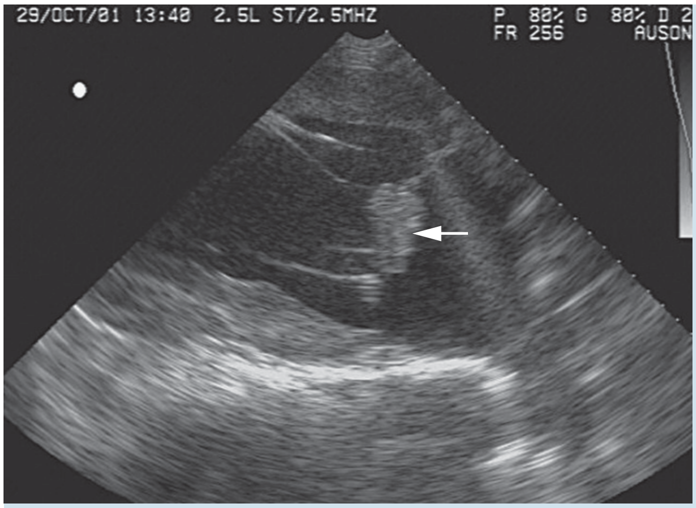


Figure 7.50: Mitral valve vegetative lesion in a 5-month-old foal that has had a history of intermittent fevers. The vegetative lesions are composed of platelets, fibrin and bacteria supported by a bed of granulation tissue that develop on the valve surface.

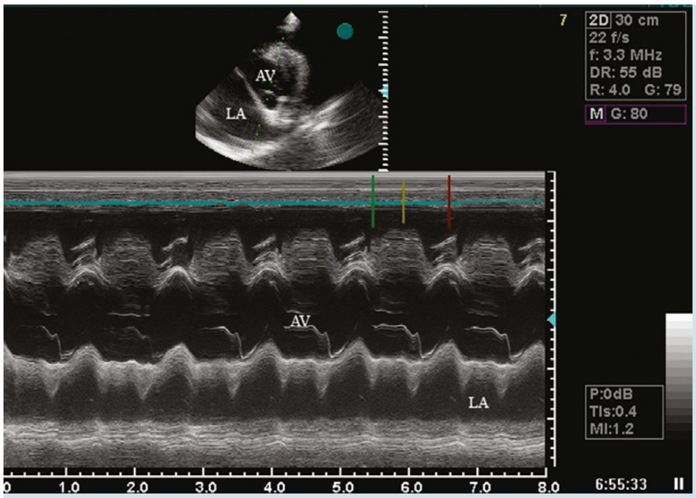


Figure 7.52: An M mode echocardiogram at the aortic level with a corresponding two-dimensional image obtained from the right fourth intercostal space from an 18 month-old Thoroughbred colt presenting with ill-thrift, depression and loud systolic and diastolic murmurs on the left side. Aortic and mitral valvulitis and fibrosis were suspected. The left atrium (LA) is dilated (AV = aortic valve).

Figure 7.51: A 7-month-old foal with a lesion similar to that in Fig 7.50 that resulted in significant mitral regurgitation. Note the small size and poor body condition of the foal.

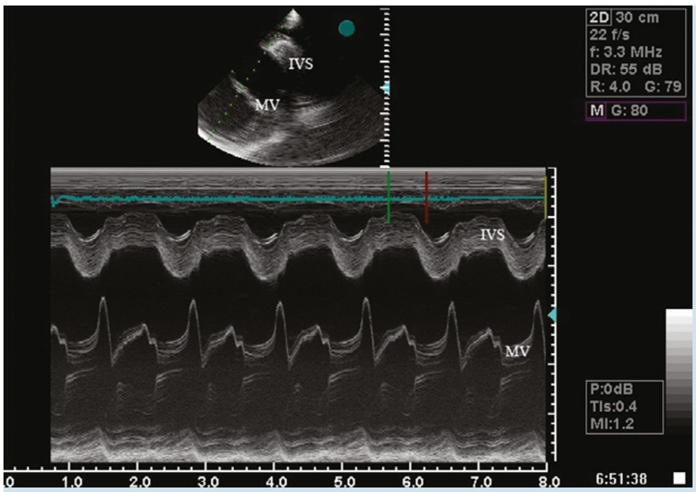


Figure 7.53: An M mode echocardiogram at the mitral valve level with a corresponding two-dimensional image obtained from the right fourth intercostal space from the same yearling as Fig 7.52. The mitral E point–septal separation is increased, reflecting left ventricular dilation (IVS = interventricular septum, MV = mitral valve).

frequently demonstrate a moderate to marked neutrophilic leukocytosis and hyperfibrinogenemia.

Treatment

Initial treatment consists of broad-spectrum antimicrobials (ideally selected based on sensitivity testing). Treatment is normally required for 4–8 weeks. Bacteremia is considered continuous but the number of bacteria at any given time may fluctuate. It is advisable to collect three blood cultures one hour apart before antimicrobial therapy is started.

Prognosis

Prognosis for left-sided vegetative valvular endocarditis is often poor. Right-sided lesions may have a slightly better prognosis (guarded) depending on the number and size of lesions, the degree of valvular and subsequent cardiac insufficiency present and chronicity.

Pericarditis (Figs 7.58 & 7.59)

Inflammatory disease of the pericardium occurs sporadically in the foal.

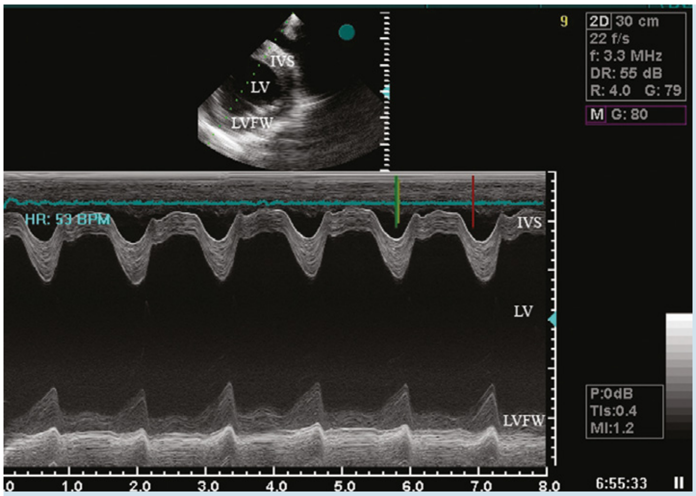


Figure 7.54: An M mode echocardiogram at the chordal level with a corresponding two-dimensional image obtained from the right fourth intercostal space from the same yearling as Figs 7.52 & 7.53. The left ventricle (LV) is markedly dilated and hyperkinetic causing exaggerated movement of the interventricular septum (IVS) and left ventricular free wall (LVFW).

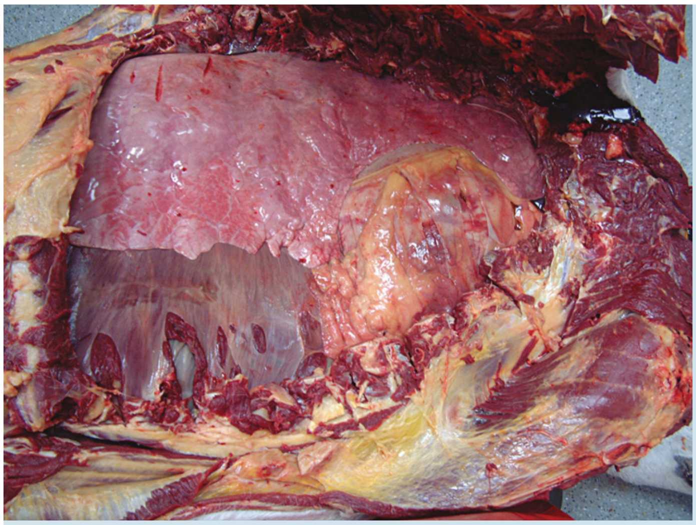


Figure 7.56: A gross postmortem specimen from the yearling in Figs 7.52–7.55 demonstrating cardiac enlargement.

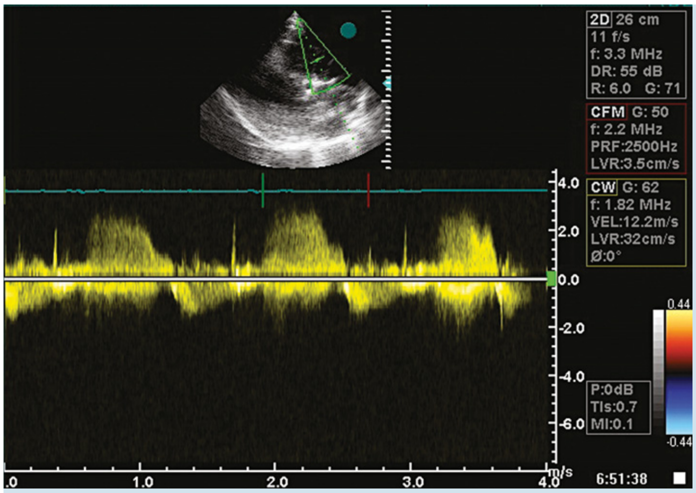


Figure 7.55: A spectral wave Doppler echocardiogram of the left ventricular outflow tract with a corresponding two-dimensional image obtained from the left fifth intercostal space from the same yearling as Figs 7.52–7.54, documenting aortic regurgitation due to valvulitis and fibrosis.

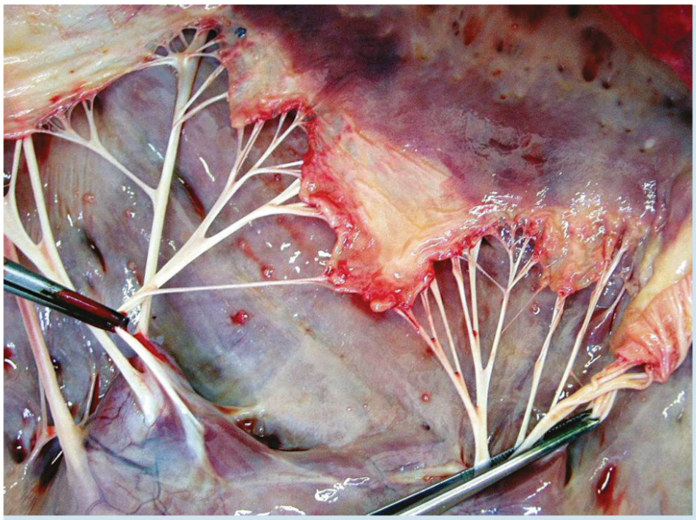


Figure 7.57: A gross postmortem specimen from the yearling in Figs 7.52–7.56. The mitral valve is thickened and distorted due to mitral valvulitis and fibrosis.

Clinical signs

- Foals typically are pyrexic, partially to completely anorexic, lethargic and may be reluctant to ambulate
- Cardiac sounds may radiate or be more muted than usual and slight ventral edema between forelegs may be observed
- A pericardial "rub" is often present during auscultation of the ventrolateral thorax

Diagnosis

Echocardiography is fundamentally diagnostic but pericardiocentesis with fluid analysis and culture may be required to identify the causal agent and its susceptibility profile. CBC results are variable, with the majority of foals demonstrating a moderate to marked neutrophilic leukocytosis with elevated fibrinogen. Animals suffering from pericardial tamponade will present with azotemia.

Treatment

- Therapy is aimed at the inciting cause with broad-spectrum antibiotics and anti-inflammatory medications initially; specific culture results may dictate longer-term treatment.
- If excessive fluid is suspected and/or cardiac tamponade is observed, echocardiography and ultrasound guided catheter or needle drainage of the pericardial space can be performed.
- Central venous pressure (if routinely monitored) >10–12 mmHg is suggestive of cardiac tamponade and should prompt further diagnostics/therapy.

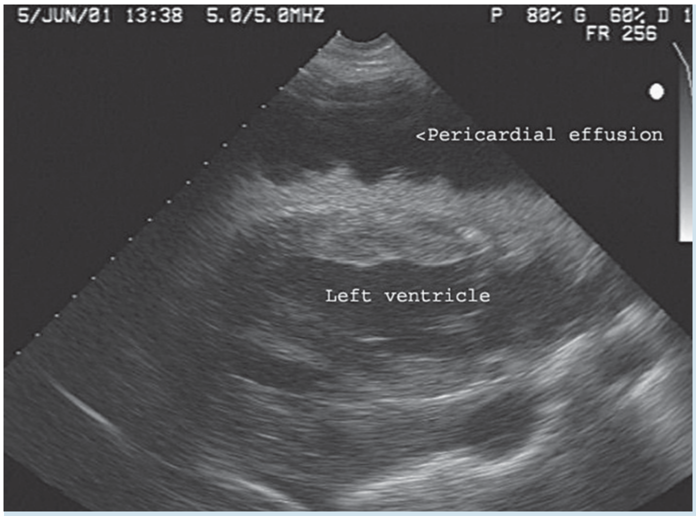


Figure 7.58: Echocardiogram from a 4-month-old thoroughbred foal with pericarditis and cardiac tamponade. A pericardial drain was placed and *Rhodoccocus equi* was isolated. Note the fibrin on the epicardial surface.

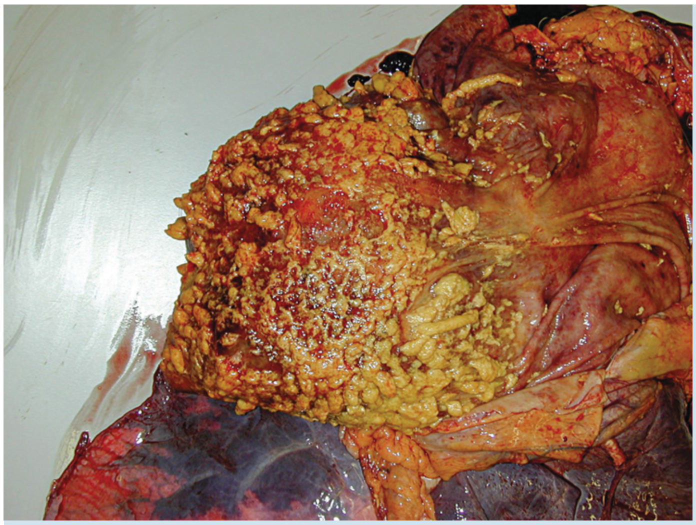


Figure 7.59: The foal in Fig 7.58 developed fibrinous constrictive pericarditis and was euthanized. Note the excessive fibrin on the epicardium (the pericardium has been removed).

Thrombophlebitis (Fig 7.60)

This is a common complication of catheter placement. Thrombosis without inflammation results in a hardened, "cord-like" vein. This arises as a result of the thrombogenic nature of the catheter or in most instances as a result of a hypercoagulable state (activation of clotting system in septicemia, disseminated intravascular coagulation, toxemia or as a result of dehydration). Thrombophlebitis results when there is concurrent infection or inflammation. This can arise as a result of poor technique during catheter placement, secondary to septicemia or from irritant drugs e.g. oxytetracycline. Potential sequelae of thrombophlebitis include:

- Unilateral vascular occlusion – limiting venous access.
- Bilateral jugular occlusion – results in impaired venous return from the head and upper neck. Such cases present with edema of the head and dyspnea as a consequence of laryngeal compression. Emergency tracheostomy may be required.
- Thromboemboli to distant organs such as heart, lung, kidney or other vascular beds occur rarely but the consequences can be grave.
- Septicemia or bacteremia could also occur.

Thrombocytopenia (Fig 7.61)

There are three main types of thrombocytopenia:

- *Reduced platelet production* may result from bone marrow aplasia or marrow infiltration due to neoplasia or severe inflammation. Although not reported this is possible in neonatal foals with severe sepsis.
- *Increased platelet consumption* such as disseminated intravascular coagulation (DIC).
- *Increased platelet destruction* such as an immune-mediated thrombocytopenia (IMTP). IMTP is either primary (idiopathic or autoimmune) or secondary, induced by drug administration, infection or neoplasia.

Clinical signs

- Thrombocytopenia results in hemorrhagic diathesis characterized by ecchymotic and petechial hemorrhages.
- Foals may show depression, lack of affinity for the dam and in some cases blood loss anemia.
- Other clinical signs include epistaxis, hematuria or spontaneous hemorrhages.

Diagnosis

- Thrombocytopenia (<100,000/μl) should be confirmed. Pseudothrombocytopenia is frequently seen as a result of platelet interaction with ethylenediaminetetra-acetic acid (EDTA) anticoagulant. This should be suspected when platelet clumping is reported on the blood smear – a second sample in 3.8% sodium citrate anticoagulant should be analyzed for comparison.
- When abnormal PT, APTT and fibrin degradation products (FDP) accompany thrombocytopenia, DIC should be suspected.
- When a normal PT, APTT, plasma fibrinogen and FDP accompany thrombocytopenia bone marrow disease or IMTP should be suspected.
- Direct immunofluorescence and flow cytometry can be used to identify surface associated Ig molecules in IMTP.
- Alloimmune thrombocytopenia should be suspected in thrombocytopenic foals in which sepsis has been ruled out and platelet antibody tests are positive.

Treatment

- No treatment exists for bone marrow hypoplasia.
- Treatment for IMTP should include:
  - Whole blood or platelet-rich transfusions are indicated in life-threatening cases.
  - Withdrawal of any medication and adjustment of antibiotic therapy (if it is required) to molecularly dissimilar agents.

A

B

Figure 7.60a & b: Distal limb gangrene secondary to thromboemboli in peripheral vascular beds. Note the marked color difference in the limbs between the gangrenous (distal) and non-gangrenous (proximal) areas.

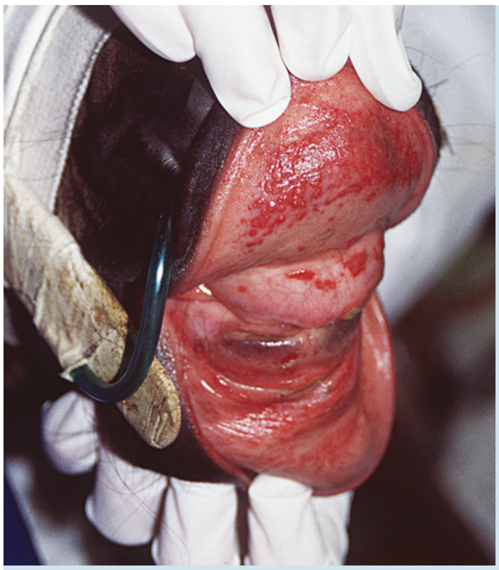


Figure 7.61: Ecchymotic hemorrhages as a result of thrombocytopenia on the oral mucous membranes.

- Identify and treat any possible underlying diseases.
- Dexamethasone (0.05–0.1 mg/kg IV q24h) should be administered with the dose decreased to 0.01 mg/kg when the platelet count is >100,000/µl. The platelet count should be normal for at least 5 days prior to withdrawal of steroid therapy.

Recommended reading

Dubin D 2000 Rapid interpretation of EKGs, 6th edn. Cover Publishing Company, Tampa

Giussani DA, Forhead AJ, Fowden AL 2005 Development of cardiovascular function in the horse fetus. Journal of Physiology 565(3):1019–1030

Marr C 1999 Cardiology of the horse. Saunders, Philadelphia

Yamamoto K, Too K 1992 Arrhythmias in newborn thoroughbred foals. Equine Veterinary Journal 24(3):169–173

CHAPTER 8

The muscular system

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Introduction

Examination of the muscular system may be difficult in animals of all ages but especially so in foals due to the large range of "normal" appearances. Determining whether a muscular disorder is present may be difficult as a variety of other conditions may have similar clinical signs thus confounding the diagnosis. History taking is particularly important especially in those conditions with a known or suspected genetic basis. Important information would include:

- duration of illness
- are the signs continuous or intermittent?
- are the signs precipitated by anything? If so, what?
- exercise
- diet
- vaccinations
- current or previous medications?
- number of affected animals
- previous family history of a muscular disorder.

Physical examination should include observation at rest, walking and trotting. Palpation of affected and apparently normal musculature should then be performed. Diagnostic tests which may be useful are estimation of serum creatine kinase (CK) and serum aspartate aminotransferase (AST), muscle biopsy, electromyography and genetic testing for certain disorders.

Serum enzyme activities

Creatine kinase (CK)

- *CK levels rise rapidly (within 2 hours) in response to even mild damage. Levels peak in 4–12 hours and decline to normal levels over 2–3 days.*
- CK is also found in CNS tissue and cardiac muscle. However, cellular damage of CNS tissue does not result in increased serum levels of CK. Isoenzyme band fractionation allows discrimination between CK of cardiac or skeletal muscle origin. However, this is rarely done when determining if a muscular disorder is present but may be performed to myocardial disease.

Aspartate aminotransferase (AST)

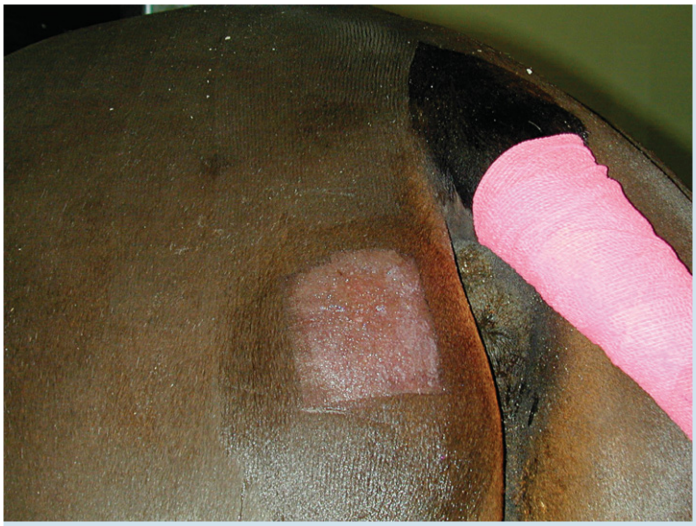
- *AST is released more slowly from muscle than CK. Levels peak in 24 hours and decline over 7–14 days.*
- Used with CK to determine if muscle damage is ongoing. Increasing AST with decreasing or normal CK indicates muscle damage has ceased.
- AST also has high activity in liver and RBC's. Liver disorders and hemolysis may result in elevations.

Lactate dehydrogenase

In older horses elevations reflect damage to skeletal or cardiac muscle or hepatic necrosis. This is not a useful enzyme in foals as it is normally elevated in growing animals.

Muscle biopsy (Figs 8.1 & 8.2)

- Prior to collecting a muscle biopsy the testing laboratory should be contacted as different staining techniques and histochemical analyses are used for diagnosing different disorders. Handling procedures vary for certain techniques and are essential to obtaining useful samples.
- Samples should be taken from diseased or abnormal muscle.
- The optimum sample for shipment is collected using an open technique. The site is surgically prepped and local anesthetic is used to infiltrate the subcutaneous tissue, not the muscle.



Figures 8.1 & 8.2: Muscle biopsy. The site is surgically prepared and subcutaneously infiltrated with local anesthetic (Fig 8.1 above) prior to a skin incision being made and a section of muscle being isolated for removal (Fig 8.2 below).

- A half-inch cube of tissue is required so a suitably long skin incision is made, followed by two parallel incisions in the muscle a half-inch apart longitudinal to the muscle fibers. Cross-secting incisions are then made to remove the cube of muscle – which should only be handled in one corner with a forceps.

Electromyography (Fig 8.3)

- Electromyography is used to detect spontaneous or evoked potentials of neurogenic or myogenic origin using electrodes positioned in the skin over a nerve or directly in the muscle.
- It can be used to distinguish denervated myofibers, motor impulses within the myofibers, abnormal electrical conductance within the myofibers or increased motor unit firing.
- It cannot directly detect upper motor neuron lesions or sensory involvement.
- Set-up costs are expensive and skill and experience are required for interpretation of results. EMG is normally only available in large referral facilities.

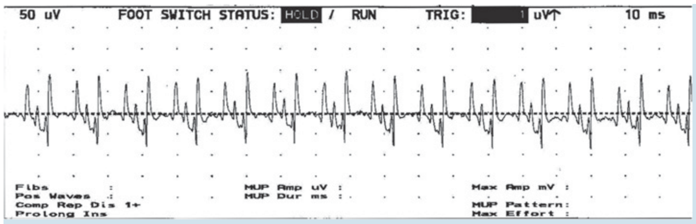


Figure 8.3: EMG from a colt with hyperkalemic periodic paralysis showing complex repetitive discharges.

Inherited / congenital conditions

Hyperkalemic periodic paralysis (Figs 8.4–8.6)

History and clinical signs

- This is a disease of Quarter horses and their crosses. To date it has only been traced to descendants of the stallion "Impressive." It is transmitted as an autosomal dominant trait.
- Most cases are 2–3-year-old well-muscled males but cases have been seen in foals as young as 4 months.
- Heterozygotes and homozygotes show clinical signs; with signs often being more severe and detected earlier in the latter.
- The most common sign is muscle fasciculations. This can be followed by muscle spasms, weakness and recumbency. Death can occur due to cardiac or respiratory failure.
- Increased respiratory rates are common during attacks. The pharyngeal and laryngeal muscles can also be affected – resulting in stridor or dyspnea.
- Attacks may be precipitated by stressors such as transport, showing, change in weather and general anesthesia.
- Hyperkalemia is common during an attack although episodes without hyperkalemia have been reported.

Differential diagnosis

Weakness, muscle fasciculations and collapse

- Generalized muscle weakness of neurological origin (e.g. equine degenerative myeloencephalopathy) or neuromuscular origin (e.g. botulism)
- Encephalitis (e.g. West Nile Virus infection) can cause muscle fasciculations
- Electrolyte disorders (e.g. hypocalcemia)
- Exertional rhabdomyolysis
- Narcolepsy/cataplexy
- Syncope
- Seizures
- Nutritional myopathy.

Figures 8.4 & 8.5: A 6-month-old foal with HYPP and myotonia congenita showing the typical well muscled appearance.

Figure 8.6: Same colt as Fig 8.5. Note the muscle contraction of the foal's left hind quarter (right side as you look at the image). This marked muscular contraction occurred following percussion.

Diagnosis

- Definitive diagnosis is by genetic testing. This can be performed on whole blood or hair root. The American Quarter horse Association will accept HYPP test results only if performed through a licensed laboratory. Testing kits for non-registration purposes are available from the AQHA.
- Diagnosed subjectively by clinical signs and signalment.
- Serum potassium levels can be measured during an attack, with levels of 6–8 mmol/L being common.
- Potassium chloride challenge testing is no longer recommended as it is difficult to interpret and can be fatal in some cases.
- Electromyography has been used for diagnosis with good reliability (90%), but its limited availability combined with the ready availability of other tests has limited its use in recent years.
- Postmortem samples that can be collected if a horse has died during a suspected attack are hair samples for DNA testing and aqueous humor for potassium concentration.

Treatment

- Mild cases:
  - Light exercise. This stimulates epinephrine release which in turn stimulates sodium-potassium ATPase activity to mobilize potassium intracellularly.
  - Feed a readily absorbable source of carbohydrates (oats). This causes insulin mediated movement of potassium across cell membranes.
  - acetazolamide (3 mg/kg orally)
- Severe cases:
  - intravenous administration of 5% dextrose with sodium bicarbonate (1–2 mEq/kg)
  - intravenous administration of 23% calcium gluconate (0.2–0.4 mL/kg) diluted in 5% dextrose (4.4–6.6 mL/kg).

Control

- Avoid feeds with high potassium content such as alfalfa and replace with timothy or Bermuda grass hay; wholegrain feeds should be fed instead of sweet feed

Dyspnea

- Obstruction of the respiratory tract (e.g. foreign body)
- Pharyngeal/laryngeal paralysis (e.g. secondary to nerve damage in strangles cases).

- Feed several small feeds daily and feeds should be given at regular intervals
- Regular exercise and ideally frequent paddock turn-out
- Avoid stressors such as rapid changes in diet, exposure to cold or over-exertion
- Acetazolamide (2–4 mg/kg q8–12hr) has been recommended to decrease the frequency and severity of episodes. The daily dose can be decreased over time until the lowest effective dose is reached.

Prognosis

The prognosis for normal activity is considered good. Counseling about breeding such horses should be offered.

Myotonia congenita (Fig 8.7)

History/clinical signs

- Myotonia congenita is a rare condition of QH and QH cross foals that results in periods of involuntary muscle contractions following stimulation or start of exercise.
- Affected animals usually show clinical signs within the first year and often have well developed musculature and a pelvic limb stiffness/lameness. The well muscled appearance is similar to that seen in HYPP affected animals.
- Only the skeletal muscle is involved in myotonia congenita, which means that progression of clinical signs is not seen beyond 12 months of age.
- A separate condition known as myotonic dystrophy has also been reported in a QH foal. This condition progresses to severe muscle atrophy and involvement of other organs.

Figure 8.7: Weanling with myotonia congenita. Again note the contracted appearance of the semimembranosus and semitendinosus muscles of the right hind. This is an older image and the animal in question has been prepared for a muscle biopsy, which is no longer recommended for diagnosis.

Diagnosis

- Frequently a tentative diagnosis can be made on the basis of breed, age and clinical signs.
- Definitive diagnosis requires EMG examination. The pathognomonic finding is described as crescendo–decrescendo, high frequency repetitive bursts with a characteristic "dive-bomber" sound.
- Muscle biopsies are not useful for diagnosis as they may be normal.

Treatment

- Most treatment modalities have poor efficacy.
- Phenytoin has been used in a number of affected horses with reported success (see Appendix 5).
- Other drugs that may be beneficial are quinine and procainamide. These are used to treat a similar condition in humans and dogs.

Prognosis

- Depends on the severity of signs but is considered poor in most cases.
- Some cases are mild and show a decrease in signs with age. Other more severely affected animals may develop extensive fibrosis with resultant restriction of movement.
- Some cases have also had concurrent HYPP.

Glycogen branching enzyme deficiency

History

This condition has emerged in recent years as an important cause of abortion and neonatal death in Quarter horses (QH) and Paint horses. It has an autosomal recessive mode of inheritance and the homozygous condition is fatal. Approximately 8% of Quarterhorses are thought to be carriers with all carriers to date tracing to the sire King P234.

Clinical signs

- Foals that survive to parturition generally present with weakness, hypothermia and severe correctable flexural deformities of all four limbs. These foals may gain strength when bottle fed or given assistance to stand and nurse.
- Recurrent hypoglycemia which may be associated with periods of collapse.
- Persistent leukopenia (4000 cells/μL).
- Elevated AST, CK and GGT.
- The progression of signs is variable with sudden death or early onset respiratory failure. Most foals die or are euthanized by 1 week of age.

Diagnosis

- The disease should be suspected in any QH or QH cross foal showing the above clinical signs.
- There are no gross postmortem changes.
- Routine hematoxylin and eosin staining of muscle may be normal or may reveal basophilic globules and eosinophilic crystalline material.

- Lack of normal PAS staining for glycogen in frozen sections of muscle.
- Genetic testing is possible at the University of California at Davis and is the most accurate means of diagnosis. Mane or tail hairs with roots intact or fetal liver tissue can be submitted for testing – this allows identification of homozygote and heterozygote horses.

Treatment/prevention

- No treatment is possible but the owners should be counseled with regard to future breeding.

Acquired conditions

Muscle atrophy (Figs 8.8–8.10)

- Muscle atrophy is a reduction in muscle size and has a variety of causes. Denervation of muscle and atrophy as a result of loss of use are both common in foals.
- Denervation of a muscle will result in >50% loss of muscle mass in 2–3 weeks. The most obvious example is "sweeney" in which the suprascapular nerve is damaged with resultant atrophy of the scapular muscles.
- Loss-of-use atrophy may be seen in foals that have been chronically lame. Limb fractures in particular can result in marked atrophy of associated muscles.

Figure 8.8: Yearling colt with recent (30 min) trauma to the base of the neck/ shoulder region following a collision with a post. Note the extensive swelling in the area.

Muscle necrosis (Figs 8.11–8.15)

History / etiology

- There are many possible causes of muscle necrosis. These include infectious, toxic, nutritional, and ischemic – but many cases are idiopathic.
- The necrosis itself may be focal or generalized. Focal necrosis is commonly seen in foals after intramuscular administration of drugs or following snake or spider bites.
- Snake and spider bites are accompanied by extensive skin sloughing.
- Muscle has a great capacity to regenerate, especially in young animals, and frequently a complete recovery is made. Extensive or severe lesions may result in fibrosis of the muscle and permanent deformity.

Clinical signs and diagnosis

- Localized swelling and pain will be evident. If associated with a limb there may be profound lameness.
- Imaging of the area affected. Radiography or ultrasonography (depending on the site) reveals soft tissue swelling and may demonstrate the presence of gas/air within the soft tissue.
- Culture any discharge from draining tracts.

Treatment

- Treatment involves supportive care, anti-inflammatories and antimicrobials in cases of infectious necrosis.

Figure 8.9: Same colt as Fig 8.8 imaged the following day. Much of the swelling has subsided but note the outward rotation of the shoulder as the horse bears weight on the affected leg (arrow). This appearance is classical of horses that have damaged the suprascapular nerve and thus have lost innervation of the muscles supporting the shoulder. Also note the way the horse is leaning towards the handler in an attempt to bear as little weight as possible on the affected limb.

Figure 8.10: Same colt as Figs 8.8 & 8.9, 4 weeks later. Note the extensive muscle atrophy of the infraspinatus and supraspinatus muscles.

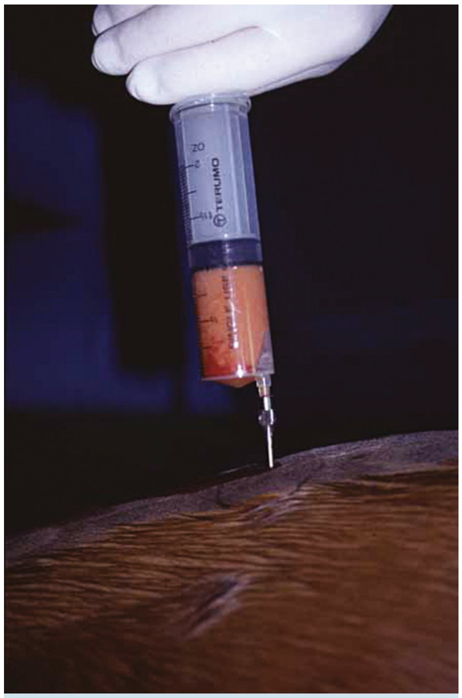


Figure 8.11: Clostridial myonecrosis. A needle aspirate is taken from a site that has been identified by ultrasonographic examination as having gas within the muscle.

Figure 8.12: Same horse as Fig 8.11. After confirmation of the presence of *Clostridium* within the aspirate, the site of infection is fenestrated.

Figure 8.13: Pigeon fever (*Corynebacterium pseudotuberculosis*) in a foal. Note the swelling of the left pectoral region and lower neck (right side as you look at the image). This is an example of infectious muscle necrosis.

Figure 8.14: Marked facial swelling in a 4-day-old foal following a snake bite.

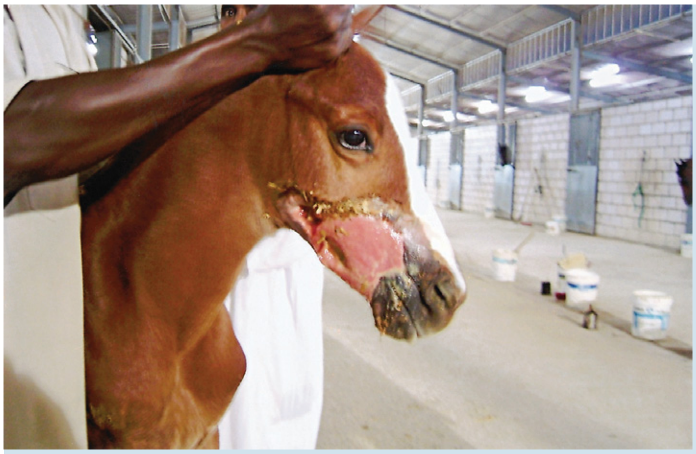


Figure 8.15: Same foal as Fig 8.13, 2 months later, demonstrating extensive necrosis of muscle and skin. Granulation tissue is present in much of the affected area.

- In cases where anaerobic infections are thought to be involved such as clostridial myonecrosis, fenestration of the area is recommended.

Nutritional myodegeneration (NMD) / vitamin E & selenium deficiency / white muscle disease (Figs 8.16 & 8.17)

History / etiology

This is an uncommon condition of young growing foals normally between 2 weeks and 7 months of age. NMD results from a dietary deficiency of selenium and/or vitamin E in gestating dams. Selenium

Figure 8.16: Recumbent weanling suffering from nutritional myodegeneration.

Figure 8.17: Postmortem specimen from the weanling in Fig 8.16, note the pale appearance of the muscle, hence giving rise to the term white muscle disease.

deficiency appears to play the most important role based on prophylaxis and response to treatment. Specific family lines show a predisposition to the condition and as such genetic factors may also play a role. The condition has a higher than normal prevalence in Fell ponies.

Two types of the disease are seen but the classification is based on the organs affected.

Skeletal or subacute form

- This form usually results in generalized muscle weakness or stiffness. It can present with asymmetric signs giving it an initial appearance of lameness and thus making diagnosis more difficult.
- Supporting muscle groups of the limbs (especially hindlimbs) may be swollen and painful.

- These foals frequently have difficulty rising and may be recumbent for prolonged periods.
- Pneumonia as a result of immunosuppression, dysphagia and aspiration, is a common sequel.
- If the diaphragm and intercostal muscles are affected respiratory distress may be present.
- Myoglobinuria may be seen in some cases.

Cardiac or peracute form

- In the cardiac form the predominant lesions are in the heart, diaphragm and intercostal muscles.
- These animals are frequently found dead or in a near dead condition. If found alive they have a rapid, irregular heart beat and profound weakness.
- Cardiomyopathy may also be seen in cases in which the diaphragm and intercostal muscles are affected.

Differential diagnosis

- Polysaccharide storage myopathy (PSSM). This is normally seen in older horses but has been reported in foals as young as 1 month of age.
- Glycogen branching enzyme deficiency. Signs are apparent at birth.
- Hypoxia induced rhabdomyolysis.
- Post-anesthetic myoneuropathy.
- Botulism

Diagnosis

- Clinical signs.
- Myoglobinuria or elevated plasma myoglobin.
- Elevated CK, AST and LDH (isoenzymes 4 and 5 are related to skeletal muscle activity and isoenzymes 1 and 2 are related to cardiac muscle activity). *Elevations are usually in the thousands IU/L. Animals which have elevations in these enzymes for other reasons rarely go past several hundred IU/L.*
- Definitive diagnosis of NMD is established by determining whole blood selenium. Normal range for selenium in whole blood is 0.07–0.10 ppm.
- Glutathione peroxidase (GSH-Px) which is selenium dependent can also be measured (20–50 U/mg of hemoglobin/min is normal in horses). Whole blood levels of GSH-Px are constant for 4–6 days at 4°C, but results should be validated against whole blood selenium.
- Vitamin E (normal >1.1–2.0 ppm) can be measured in plasma but deteriorates rapidly. Serum samples should be covered and placed on ice and analyzed immediately.

Treatment

- The subacute form may respond to treatment with vitamin E/selenium injections. Injectable selenium should be given IM at a dose of 0.055–0.067 mg/kg (2.5–3 mg/45 kg body weight). Injections can be repeated at day 3 and once again between days 8–10 after the initial treatment. Absorption and distribution occur rapidly and may account for the rapid improvement in clinical signs in some cases. Injection site reactions are common so dilution with saline and dividing the dose between two pectoral sites is recommended. Avoid using the neck as an injection site as pain from injection reactions may prevent nursing. Vitamin E should also be supplemented in either injectable or oral form. The latter is easier to administer and contains 500 IU/mL, the published recommended dose for NMD is 2–6 IU/kg, however the editors recommend using up to 20 IU/kg.
- Supportive care and rest are important aspects of treatment.
- Many cases are advanced or have extensive muscle fibrosis when diagnosed and respond poorly to treatment.
- Those with the cardiac form that are diagnosed and treated early usually respond poorly and die within 24 hours.
- It is imperative that pregnant mares receive adequate vitamin E in the last trimester of pregnancy and a nutritionist should be consulted to formulate an appropriate diet.

Figure 8.18: Weanling with immune-mediated myositis as a result of *S. equi* infection. Note the generalized muscle atrophy.

Immune-mediated myopathy associated with *Streptococcus equi* infection (Fig 8.18)

History / etiology

- Some horses including foals that have been exposed to infection with *Streptococcus equi* have developed myositis with rapid atrophy of the lumbar and gluteal muscles.
- The M protein of some streptococcal organisms has the same amino acid sequences as the contractile protein myosin with a resultant immune-mediated myositis in some animals upon infection with these organisms.
- The affected horses may not have any concurrent signs of strangles.

Diagnosis

- Clinical signs of rapid muscle loss with a history of exposure to strangles.
- Elevations in serum CK and AST.
- Muscle biopsy. Common findings on biopsy are lymphocytic vasculitis, atrophy of Type 2 fibers, regeneration and fibrosis.

Treatment

- Antibiotic therapy should be used if concurrent signs of strangles are present such as lymphadenopathy or leucocytosis.
- Immunosuppressive drugs such as prednisolone (1 mg/kg for 7–10 days followed by tapering doses over 1–2 months) or dexamethasone (0.1 mg/kg orally for 7–10 days followed by tapering doses over 1–2 months) are required to temper the host's immune response.

Post-anesthetic myoneuropathy (Fig 8.19)

This disorder is not usually seen in young foals but may be seen in older foals and weanlings. There are two different forms, of which the generalized form is rarely encountered in young horses and is not discussed here.

Localized myopathy-neuropathy

- This is usually seen in muscles that are in contact with a hard surface during anesthesia or those in which arterial blood supply is diminished.
- Affected muscles depend on the positioning of the animal during anesthesia. Those in dorsal recumbency may have the gluteals affected. Those in lateral recumbency may have the deltoid, triceps, masseter or hindlimb extensors affected. Local nerves may also be affected, resulting in a temporary paralysis.
- Affected muscles are swollen, hot and painful and the animal may be reluctant to bear weight on an affected limb.

Diagnosis

History, clinical signs and elevations in CK and AST are usually enough to diagnose the condition.

Figure 8.19: Post-anesthetic myoneuropathy in a weanling filly following colic surgery. Note how the right hindquarter is elevated above the left. The right gluteal muscles were rigid and painful to touch.

Treatment

- Supportive care and anti-inflammatories are usually sufficient for mild cases.
- More severe cases may require fluid therapy, DMSO and dantrolene (2–4 mg/kg PO q24 h).

Rupture of the peroneus tertius (Figs 8.20 & 8.21)

History

The peroneus tertius is an entirely tendinous muscle that plays an important part of the reciprocal apparatus of the hindlimb, coordinating flexion of the hock and stifle. Rupture of the tendon or avulsion of the origin of the tendon can occur in young foals and in some cases can occur at foaling as a result of dystocia.

Clinical signs and diagnosis

- The clinical presentation is pathognomonic as disruption of this tendon allows extension of the hock while the stifle is flexed.
- Ultrasonography can be used to assess the extent of disruption.

Treatment

Prolonged box rest (3 months) frequently results in complete recovery in adult horses. The outcome may not be as satisfactory in young foals and future athletic ability may be compromised.

Figure 8.20: Rupture of the peronius tertius. Note the classical extension of the hock.

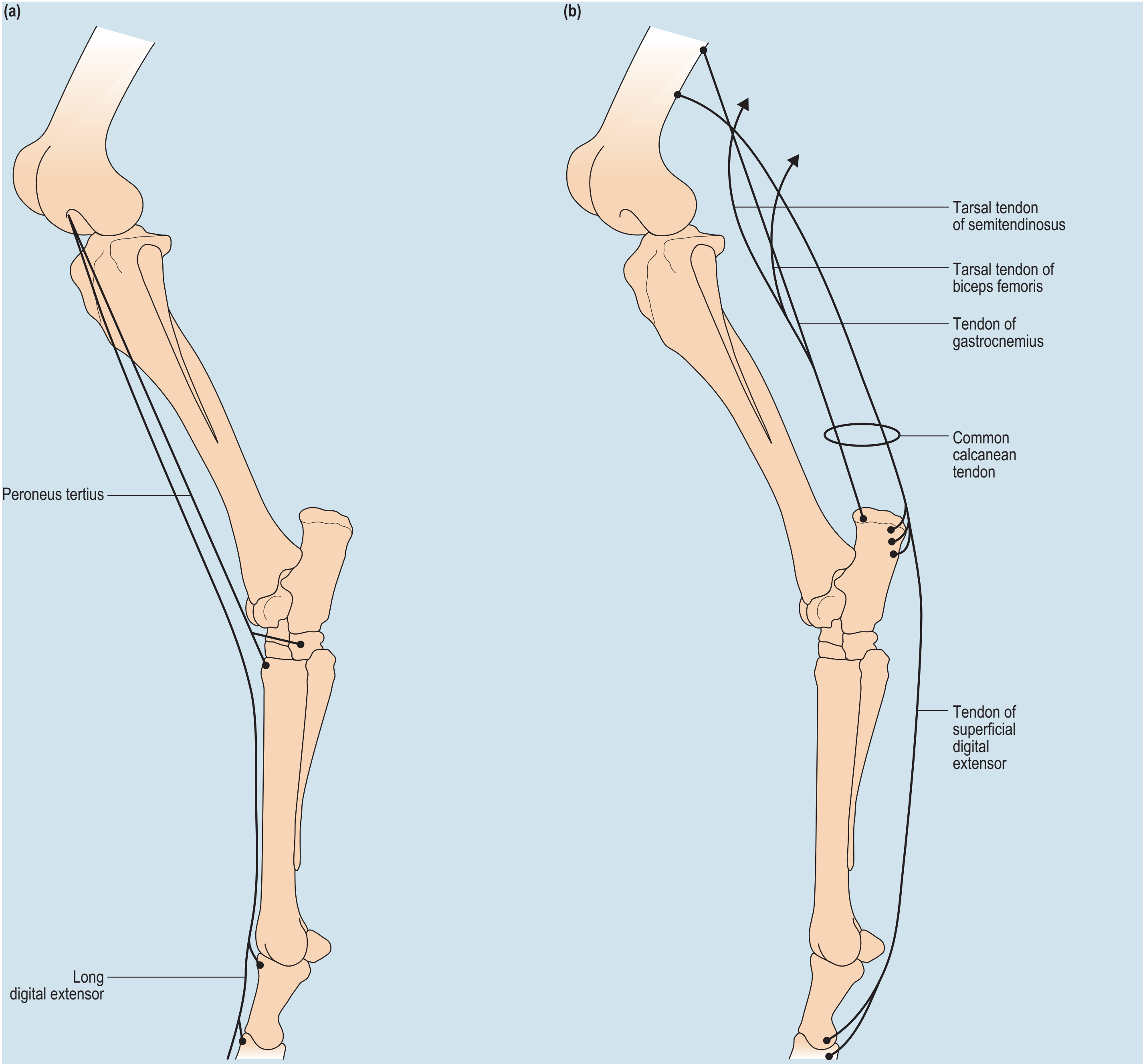


Figure 8.21: Schematic representations of the anatomy of the hindlimb showing the origins and insertions of the peroneus tertius and gastrocnemius. (a) The ***peroneus tertius*** arises (by a common tendon with the long digital extensor) from the lateral condyle of the femur and, passing cranial to the tibia, ends by complex attachments on certain tarsal bones and the proximal end of the large metatarsal bone. (b) The ***gastrocnemius*** arises from the supracondylar tuberosities on the caudal surface of the femur and ends as part of the common calcanean tendon on the calcanean tuber.

Gastrocnemius muscle rupture (Figs 8.22–8.27)

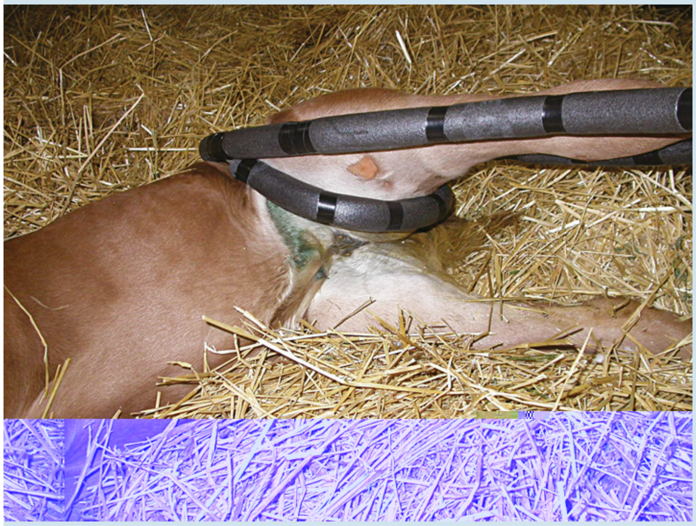
History

Rupture of the gastrocnemius muscle can occur in young foals as a result of overextension while chasing the dam or as a foaling injury. It can also occur secondary to severe flexor tendon laxity or tarsal contracture.

Clinical signs and diagnosis

Rupture of this muscle results in a typical crouched stance and inability to bear weight on the affected limb. The gastrocnemius may be markedly swollen secondary to hemorrhage. It is not uncommon to see marked anemia in affected foals. Foals have died acutely secondary to a severe gastrocnemius rupture and hemorrhage.

Figures 8.22 & 8.23: Rupture of the gastrocnemius. Note the extensive swelling and the dropped appearance when standing (different foals).



Figures 8.24–8.26: Fitting of a well padded Thomas–Schroeder splint. This splint limits excessive motion within the cast, because of the encircling portion of the splint in the inguinal region as well as the distal extension encircling the foot.

Figure 8.27: The Thomas–Schroeder splint can stop further injury and the potential for hemorrhage as well as allow the ruptured muscle ends to be in close apposition.

Figure 8.28: Pressure sores are not uncommon on the distal tibia, calcaneus and tuber coxae. In this case the splint was removed after 5 weeks and stall rest was continued for a further 4 weeks. This foal was considered sound enough to go into training as a 2-year-old.

Treatment

- Treatment most commonly involves placing a Thomas–Schroeder splint for 1 month while confining the foal to stall rest. Following removal of the splint, box rest is required for a further month.
- Care should be taken in constructing the splint to ensure it is a correct fit for the foal in question.
- Careful padding and regular re-assessments are required to ensure that pressure sores (Fig 8.28) do not develop.
- Tube casts can also be used but must be changed frequently and cast sores are inevitable.

Prognosis

The prognosis for athletic function is guarded.

Recommended reading

Dyson SJ 2003 Other soft tissue injuries. In: Ross MW, Dyson SJ (eds) Diagnosis and management of lameness in the horse. Saunders, Philadelphia, p 708–711

Santschi EM 2004 Musculoskeletal disorders of foals. In: Reed SM, Bayly WM, Sellon DC (eds) Equine internal medicine. Saunders, Philadelphia, p 1431–1440

CHAPTER 9

The skeletal system

Dwayne Rodgerson DVM, DACVS

Inherited / congenital conditions

Deformities of the vertebral column (Figs 9.1–9.4)

Deformities of the vertebral column generally involve the cervical (wry neck) and lumbar (scoliosis) regions. Conditions within the cervical vertebral column are more common. The abnormality within the vertebral column can be due to lack of vertebra, malformed vertebra, or fused vertebra. In utero, a severe cervical vertebral deformity can predispose a mare to dystocia due to the inability of the foal's head to engage the pelvis.

The etiology of vertebral deformities is often unknown. Failure of proper embryologic development of the vertebral column may be associated with possible genetic defects. Abnormal in utero positioning has also been proposed.

Diagnosis

- Visual examination
- Digital examination
- Radiology – helps confirm and further evaluate the degree and severity of the deviation
- Postmortem examination.

Treatment

- Conservative management:
  - mild cases can potentially survive and live without significant gait problems
  - severe cases are generally humanely destroyed
- Severe cases of cervical vertebral deformities that cause dystocia will either require a fetotomy or cesarean section to save the mare.

Polydactyl (Figs 9.5–9.8)

The presence of an extra digit is uncommon in the horse but numerous case reports have been published describing the presence of an extra rudimentary digit. The rudimentary digit most commonly is located at the level of the metacarpo-phalangeal or metatarso-phalangeal joint. The digit may vary from a small enlargement of the distal second/fourth metacarpal/metatarsal bones to an actual small isolated digit with a small hoof. Though uncommon there are cases where the rudimentary digit is located at the level of the carpus.

Diagnosis

- Visual examination
- Digital examination
- Radiology.

Treatment

Treatment is usually by surgical removal.

- Surgical removal of the extra digit is for cosmetic reasons and to prevent potential interference injuries.

Figure 9.1: Foal with scoliosis. The curvature of the spine can be seen by following the dorsal stripe.

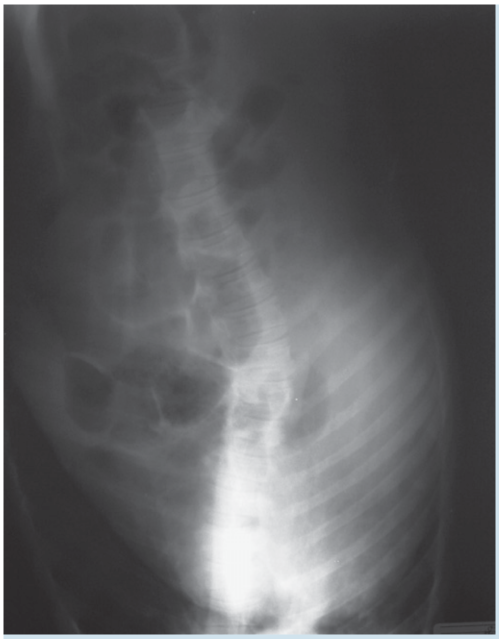


Figure 9.2: Radiographic image of foal in Fig 9.1 showing obvious curvature (scoliosis) of the spine.

Figure 9.3: Weanling with kyphosis.

Figure 9.4: Skeletal deformities can affect areas other than the vertebral column. The foal imaged here has obvious curvature of the head.

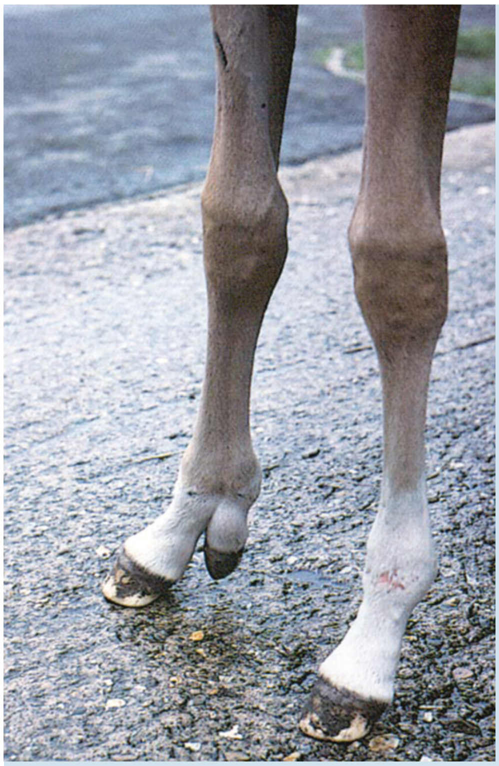


Figure 9.5: Polydactyl with the extra digit located at the level of the metacarpo-phalangeal joint.

Figure 9.6: Polydactyl with the extra digits making contact with the ground.

- The surgical removal of an extra digit is aimed at removing the digit at the level of the normal location of the distal metacarpal/metatarsal bones.
- Special attention is paid to ensure proper ligation of the rudimentary digital arteries associated with the digit.
- For cases involving polydactyl at the level of the carpus, surgical removal is aimed at surgically transecting the digit close to the proximal margin of the small metacarpal bones.

Figure 9.7: Dorso-palmar radiograph of polydactyl.

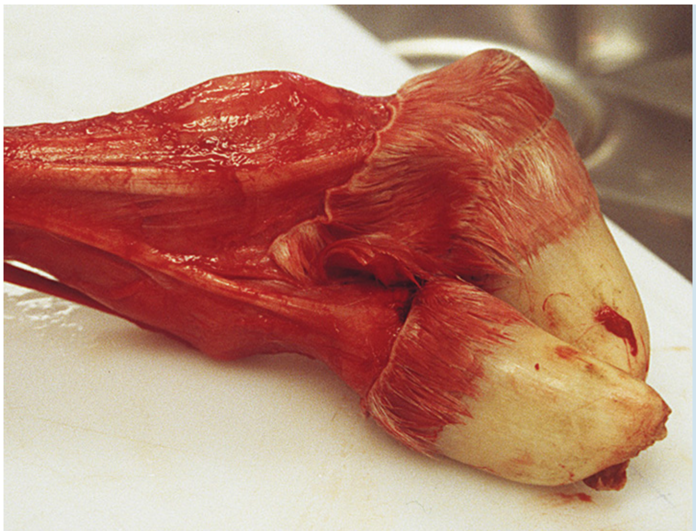


Figure 9.8: Postmortem specimen of polydactyl.

Windswept (Figs 9.9–9.12)

Foals with a "windswept" conformation have both forelimbs and/or both hindlimbs having an angular limb deformity in the same direction. For example, a foal would have a carpal valgus deformity in the left forelimb and a carpal varus in the right forelimb. Generally this type of conformation affects the carpus or tarsus more than the fetlocks. This type of conformation has often been attributed to uterine positioning and soft tissue laxity. The severity of the deviations is most

Figure 9.9: Severely windswept foal. Note the angular limb deformities in the same direction; i.e. a tarsal and metatarsal varus in the right hind and a tarsal and metatarsal valgus of the left hind limb. Note also the flexor laxity of the forelimbs.

severe after birth and will markedly improve over the first 14–21 days of the foal's life.

Diagnosis

- Visual examination
- Radiology may be used to further assess the degree of valgus or varus deformity involved.

Figure 9.10: Same foal as Fig 9.9 imaged at 10 weeks of age. A periosteal elevation of the medial aspect of the right hind fetlock was performed in conjunction with corrective trimming.

Treatment

Conservative management

- Limit exercise
  - stall rest to small pen exercise for 2–4 weeks
  - most cases improve greatly in 2–3 weeks
- Radiology – to ensure proper ossification of carpal and tarsal bones
- Foot care – generally begin around 2–3 weeks depending on maturity of feet
  - trimming: gently lower/trim the inside of the foot for a varus deformity, gently lower/trim the outside of the foot for a valgus deformity
  - shoeing/extensions: extension placed on the inside of the foot for a valgus deformity, extension placed on the outside of the foot for a varus deformity.

Surgical management

This is reserved for cases which are severe.

- Hemicircumferential periosteal transection and elevation
- Transphyseal bridging:
  - transphyseal staples
  - screws and wires
  - single transphyseal screw.

Figure 9.11: Same filly as Figs 9.9 and 9.10 imaged as a yearling. Note the mild curvature of the cannon bone of the right hind limb.

Figure 9.12: Windswept foal with flexor laxity of the hindlimbs. This foal had difficulty standing.

Congenital contracted tendons (Figs 9.13–9.18)

Congenital flexural deformities are believed to occur due to uterine malpositioning, genetic defects, and toxic or infectious insults during pregnancy; recently, congenital hypothyroidism has been identified as a cause. There is a discrepancy between the relative lengths of bone and soft tissue. Clinical signs consist of carpal flexion, fetlock flexion, or deep digital flexor tendon contraction depending on the structures affected:

- superficial flexor contracture results in an upright distal limb but a normal hoof ground contact
- deep flexor contracture results in an upright "on the toe" posture
- combined contracture results in an upright pastern and upright hoof.

The condition can occur alone or can be seen with joint contracture.

Diagnosis

- Visual examination
- Digital examination may be necessary to differentiate tendon contracture from joint contracture.

Treatment

The type of treatment undertaken depends on the severity of the contracture.

- Correction in some foals is spontaneous with restricted exercise. This is most common in foals that have mild contracture with either normal hoof ground contact or only a slightly elevated heel. Restricted exercise should consist of paddock turn-out for 30 minutes 2–3 times per day or in hand walking on a hard surface for 5–10 minutes up to 4 times per day. Foals with a slight elevation of the heel generally respond well to a single dose (1–3 g) of oxytetracycline with or without support wraps.
- Physiotherapy in the form of gentle manipulation of the limb into a normal position 4–6 times per day may be beneficial.
- Moderate deformities may respond to oxytetracycline; a total dose of 1–3 g IV, up to 2–4 injections 24–48 hours apart. This can be used for foals which are standing on their toe but not knuckling forward onto the dorsal hoof wall. A response is usually seen in 24–48 hours. The treatment may be contraindicated in foals that have laxity of other limbs. If the response is not sufficient the treatment should be combined with the application of splints.
- In more severe cases various types of splints and casts can be used to help correct the contracture. These are essential for foals in which the contracture results in knuckling forward onto the dorsal hoof wall. This treatment is combined with the administration of intravenous oxytetracycline. If splints and casts are used, ample padding and frequent changes are needed to prevent pressure sores. The frequency of changes depends on the severity of the contracture.

Figure 9.13: Mild congenital tendon contracture that responded to bandaging and intravenous oxytetracycline.

Figure 9.14: Congenital tendon contracture of a single forelimb. This responded to application of a splint (see Fig 9.15).

Many severely contracted foals may be unable to stand without the aid of splints but also may experience great pain with the manipulation of the limb into a normal position by the splint. Such foals may be seen to hyperventilate and the administration of non-steroidal anti-inflammatories combined with anti-ulcer medication should be administered. These foals will need frequent "breaks" (2–4 hours on then 2–4 hours off) from their splints. Foals that are not so severely affected can remain in their splints for longer periods (up to 8 hours). It may be best for the veterinarian to apply the splints during morning examinations and then have the splints removed in the evening, especially if there is no "night-watch" service.

Figure 9.15: This image demonstrates the making of a splint by using the normal leg as a template. The normal limb is bandaged and cast material then applied. The cast is then cut and applied to the affected limb which is similarly bandaged. A full or half cast can be used on the affected limb. Casts can be designed with or without the hoof enclosed. If the hoof is not enclosed some other method of extending the toe such as taping should be used. This method of splint production, although time consuming, ensures that the splint applied to the foal is a correct fit.

- Surgical intervention is generally not necessary but in some severe carpal contractures, surgical transection of the ulnaris lateralis or carpal fascia has been helpful.

Congenital joint contracture (Figs 9.19–9.25)

Carpal and fetlock joint contracture is an uncommon condition believed to involve contracture of the joint capsule. In most cases the joint capsule shows fibrosis in a severe flexed position. The condition is usually unilateral, but can be bilateral.

Etiology is believed to be similar to congenital contracture of the flexor tendons including intrauterine malpositioning, genetic defects, and toxic or infectious insults during pregnancy. Severe contractions greater than 90° have a poor prognosis.

Diagnosis

- Visual examination – it may be difficult to distinguish joint contracture from tendon contracture
- Digital examination of the limb does not reveal significant tension of the flexor tendons
- Radiology.

Treatment

- Generally the degree of contracture is severe and foals do not respond well to medical management

Figure 9.16: (a) Severe congenital flexural contracture in a 12-hour-old foal. This foal was unable to stand without support. The foal had splints applied to both limbs and received intravenous oxytetracycline. The splints in this case were used on a 4 hours on / 4 hours off basis. (b) Same foal 24 hours later. An improvement can be seen in that the foal is now able to support himself on the point of his toe. (c) Same foal 48 hours after the first image. A significant improvement can be seen at this stage with normal weight bearing. The use of the splints was discontinued at this point.

Figure 9.17: A commercial splint boot.

Figure 9.18: A commercial Dyna^{TM} splint. This can be used to treat either tendon or mild metacarpophalangeal joint contracture.

Figure 9.19: Severe bilateral congenital contracture of the carpal joints. Such contracture frequently results in dystocia as the forelimbs cannot be extended for delivery. This foal was euthanized.

Figure 9.20: Congenital contracture of the carpal joint of the right forelimb. Note the limb cannot be manipulated into a normal position.

Figure 9.21: Congenital contracture of the tarsal joint of the right hindlimb. The foal was unable to rise without assistance.

A

B

Figure 9.22: (a) Same foal as in Fig 9.21 in lateral recumbency. Note the limb cannot be manipulated into a normal position. (b) Radiographic image of the tarsal joint. Note the greater than 90° flexion of the joint.

- Surgical resection of the fibrosis can be attempted but is generally unsuccessful
- Humane destruction of the foal is recommended
- Severe cases may result in dystocia that requires a fetotomy or caesarian section to save the mare.
- Mild cases, in which weight can be borne on the limb, can be treated with a regime of bandaging and controlled exercise.

Flexural laxity (Figs 9.26–9.28)

Tendon laxity or weakness in newborn foals is not uncommon. It is due to musculotendinous weakness, which can result from immaturity, systemic illness, lack of exercise or excessively long toes.

Clinically, the foal walks on the caudal aspect of the foot and the toe of the hoof “rocks-up.” Generally the condition occurs in rear feet or all four legs.

The laxity can occur at the distal interphalangeal joint, metacarpo- or metatarso-phalangeal joint, carpus, and tarsus.

Diagnosis

- Visual examination
- Digital examination.

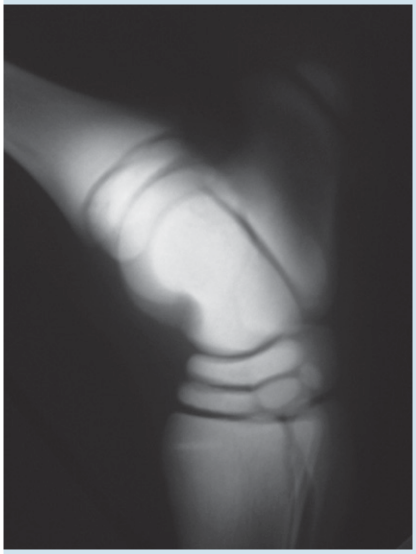
Treatment

- Controlled exercise is to be encouraged in mild cases of laxity in newborn foals. Turn-out in a small paddock for 30–60 minutes 2–3 times per day may result in a marked improvement in 2–3 days.
- In severe cases the hoof is supported with heel extensions. In general bandaging is contraindicated as it results in relaxation of the soft tissues and further laxity. However, in very severe cases *small, light* bandages may be necessary to protect the palmar/plantar surfaces of the fetlocks.
- Older foals may have the hooves trimmed so that the foal has a flat weight-bearing surface.

Congenital luxation of the patella (Figs 9.29 & 9.30)

Congenital luxation of the patella is rarely seen in foals and when observed is most commonly in miniature horse foals. The condition may be unilateral or bilateral and can be an intermittent luxation to

A



B

Figure 9.23: (a) Normal leg from the same foal as Fig 9.21. Note that this limb can be extended normally. (b) Radiograph of the limb in a neutral position.

Figure 9.24: Congenital contracture of the metatarso-phalangeal joint. In mild cases such as this where the joint is contracted but weight can still be borne on the hoof, conservative management in the form of bandaging and controlled exercise yields good results.

Figure 9.25: Bilateral congenital contracture of both metatarso-phalangeal joints. More severe cases such as this require splinting with intravenous oxytetracycline. The decision to administer oxytetracycline in mild cases of tendon contracture may be influenced by the pastern angle of the unaffected limbs. It is not unusual to see a foal with a flexor contracture in one or two limbs and flexor laxity of the other limbs.

Figure 9.26: Moderate flexor laxity.

(a)

(b)

Figure 9.27: (a) Severe flexor laxity. (b) Same foal following the application of commercially available heel extensions. Note the improvement in the pastern angle. However a longer heel extension such as that in Fig 9.28 would have been more suitable for this foal.

a complete luxation that is difficult to reduce manually. Most commonly the luxation is in a lateral direction.

Foals with intermittent luxation may show intermittent lameness and snapping action over the point of the stifle. Foals in persistent lateral luxation have a flexed stifle and flexed hock appearance due to the equine reciprocal apparatus. Foals with bilateral luxation have difficulty standing, but if standing, foals will often carry their pelvis lower then the lumbar spine.

Etiology of the congenital luxation is often associated to dysmaturity of the trochlear ridges (most commonly the lateral trochlear ridge), and soft tissue laxity associated with the femoro-patellar joint.

Figure 9.28: Heel extensions in place. The length of the heel extension depends on the severity of the laxity. The length can be shortened as the foal improves. Care should be taken with heel extensions that encase the hoof as they inhibit lateral hoof growth and can result in hoof deformities if left in place too long (>10 days).

Figure 9.29: Bilateral congenital patellar luxation. This foal could not stand without assistance.

Diagnosis

- Visual and digital examinations of the stifle are often all that is needed to diagnose patellar luxation.
- Radiographs will help to confirm the disorder and identify the cause of the patellar luxation:

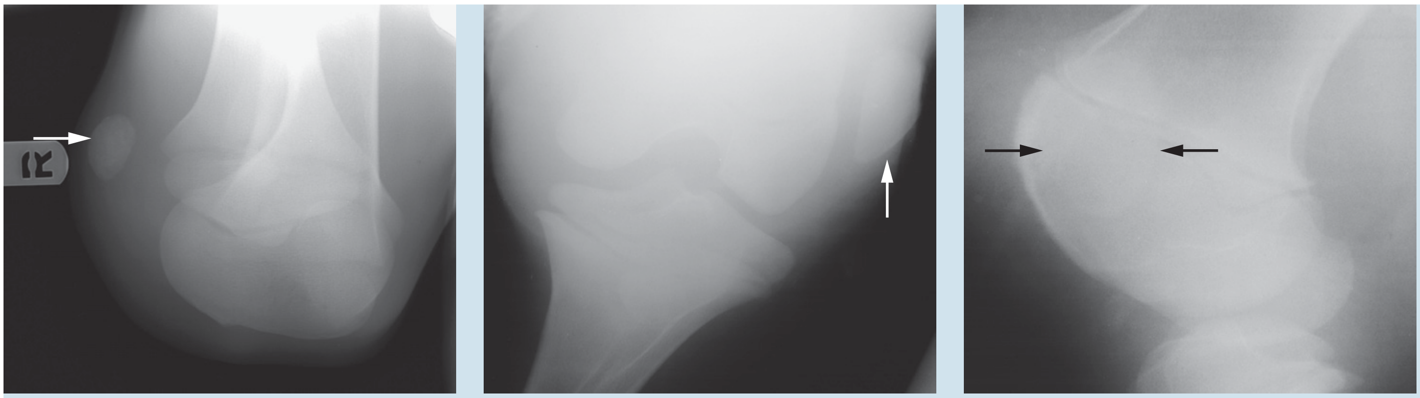


Figure 9.30: Skyline, caudo-cranial and lateral radiographic images of the foal in Fig 9.29. Note the lateral position of the patella (arrows) in all images.

- Lateral radiographs will reveal a patella in a more caudal position than normal overlapping the trochlear ridges.
- caudal to cranial radiographs reveal the patella in a more lateral (most common) or medial position. In lateral luxations, the patella is positioned lateral to the lateral condyle
- it is imperative to take a skyline view of the distal dorsal femur to evaluate the trochlear ridges.

Treatment

- Medical management of patellar luxation is reserved for intermittent cases. Exercise restriction and manual replacement of the patella is performed with the expectation of soft tissue laxity improving and eliminating episodes of patellar luxation.
- Surgical management of patellar luxation is recommended for cases with more persistent luxation:
  - if the trochlear ridges are within normal limits, surgical stabilization of the patella is recommended; contralateral release of the soft tissue supporting structures can also be performed to help maintain the patella in a normal position
  - for lateral luxation of the patella, medial imbrications and lateral release of the soft tissue has been well described
  - if the trochlear ridges are hypoplastic and there is minimal depth to the trochlear groove, trochleoplasty is recommended
  - in severe cases, all three procedures may be required.
- Prognosis for unilateral cases of patellar luxation should be guarded to fair and guarded for bilateral cases.

Common digital extensor tendon rupture (Figs 9.31 & 9.32)

Rupture of the common digital extensor may be seen as a primary condition in foals or secondary to flexural deformities of the carpal or metacarpo-phalangeal joints that result in increased tension in the tendon.

Clinical signs and diagnosis

- Most affected foals have a normal stance but a slight over-at-the-knee appearance is also common. Occasionally foals may knuckle forward on the fetlock.

Figure 9.31: Foal with bilateral rupture of the common digital extensor tendon knuckling forward onto the dorsal surface of the fetlock joints.

- There is a characteristic fluctuant swelling over the dorsolateral aspect of the carpus at the level of the distal carpal joints.
- Digital palpation reveals fluid distension of the tendon sheath and the ruptured ends of the tendon can usually be identified.
- Ultrasonography can be used to confirm the diagnosis.

Treatment

- Foals with primary rupture (no associated flexural deformities) should be box rested and have bandages applied to prevent abrasion of the dorsal aspect of the fetlock.
- Foals that frequently knuckle forward onto the fetlock or have concurrent flexural deformities or cuboidal bone malformation should have a splint applied to the palmar aspect of the limb from

Figure 9.32: Unilateral common digital extensor tendon rupture. Note the swelling at the level of the distal carpal joint (arrow).

the elbow to the fetlock. It is usually necessary for the foal to wear splints for 2–4 weeks. In that time the splints and associated padding should be changed frequently to avoid complications. Bandaging for several weeks is recommended after splint removal to maintain pressure on the area of rupture in order to decrease the resulting blemish.

Tarsal collapse and incomplete ossification (Figs 9.33–9.42)

Tarsal collapse can occur in premature and young foals. Predisposing factors include dysmaturity, trauma, and the incomplete ossification of the tarsal cuboidal bones. Incomplete ossification of the tarsal/carpal cuboidal bones can be classified into four groups:

- Grade 1 – some cuboidal bones with no ossification
- Grade 2 – all cuboidal bones had radiographic evidence of some ossification
- Grade 3 – cuboidal bones are small and round with consequent "wide" appearance of the joint spaces
- Grade 4 – cuboidal bones are shaped like the adult counterparts.

Foals with tarsal bone collapse will show weakness through the tarsus and often may show swelling over the plantar aspect of the proximal third metatarsus (curby appearance). Foals can show minimal to moderate signs of lameness.

Diagnosis

- Visual examination will allow a presumptive diagnosis.
- Lameness examination should be performed to rule out other conditions. Incomplete ossification and tarsal collapse are normally associated with minimal lameness.
- Digital examination. Digital palpation over the dorsal aspect of the distal tarsus may elicit signs of pain. It is very important to ensure sepsis is not present.
- Radiology will not only allow assessment of the degree of ossification of the carpal/tarsal bones but will also reveal collapse of the cuboidal bones if present.

Treatment

- Foals should be restricted from excessive exercise to prevent compression of the tarsal/carpal cuboidal bones.
- The tarsus/carpus should be radiographed every 10–14 days.
- Foals should be slowly introduced to normal turn-out even after complete ossification has occurred.
- Ensure the foal is getting adequate nutrition.
- If sepsis is suspected based on blood work and radiographic findings, foals should also be started on broad-spectrum antibiotics.

Acquired conditions

Acquired contracted tendons (Figs 9.43–9.45)

Acquired flexural deformities can occur following a traumatic insult to the affected limb which causes the foal to protect and not bear weight evenly on both limbs. The limb which is injured can potentially start to show both fetlock and carpal contracture. Injuries to the coffin bone can result in foals developing distal interphalangeal joint contracture.

Acquired flexural deformities can also occur due to growth discrepancies between the normal longitudinal bone growth and the flexor tendon elongation. Foals which are developing rapidly or being fed excessively can develop both fetlock and carpal contracture. Foals growing rapidly will often show evidence of very mild carpal contracture (over at the knees) multiple times during the first 6–12 months of age. These cases respond well to managing the foal's energy and protein intake.

Deep digital flexural contracture presents with the hoof having a raised heel or "club foot" appearance. The deep digital flexor tendon is very tense. Two types of contracture are documented:

- Type I is when the dorsal surface of the hoof wall is less than 90 degrees
- Type II is when the dorsal surface is greater than 90 degrees.

Treatment consists of making necessary dietary changes (grass hay and ration balancing), exercise to stretch the tendon or complete rest to relax the muscle, hoof trimming to lower the heel, and possibly a toe-extension. Surgical management involves inferior check ligament desmotomy (most common), which should be performed as early as possible and prior to 5 months of age for best results. Deep digital flexor tenotomy is performed less commonly and is usually reserved for cases that have failed to respond to other forms of treatment.

Fetlock contracture presents with either a very upright pastern or the fetlock knuckling forward with a normal hoof angle. Tendon involvement is complex and variable, and in most cases contraction of both the SDF and DDF tendons are present. Treatment consists of dietary changes to decrease the caloric intake. Exercise should be

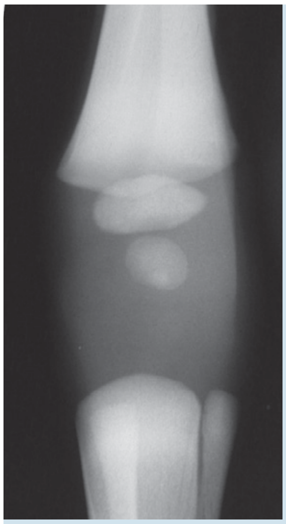


Figure 9.33: Grade 1 incomplete ossification of the carpal joint. Grade 1 is assigned when some of the cuboidal bones of the carpus and tarsus have no radiographic evidence of ossification.

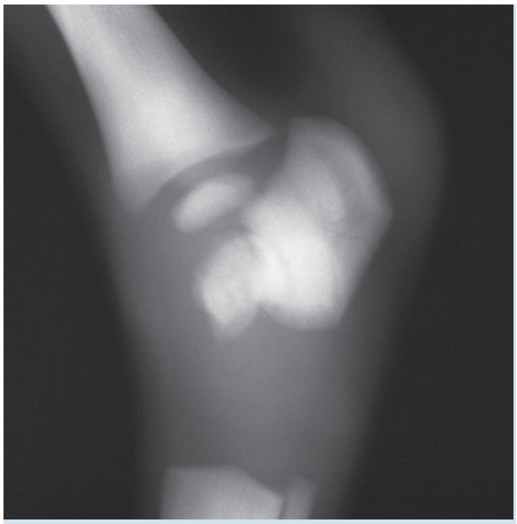


Figure 9.34: Grade 1 incomplete ossification of the tarsal joint.

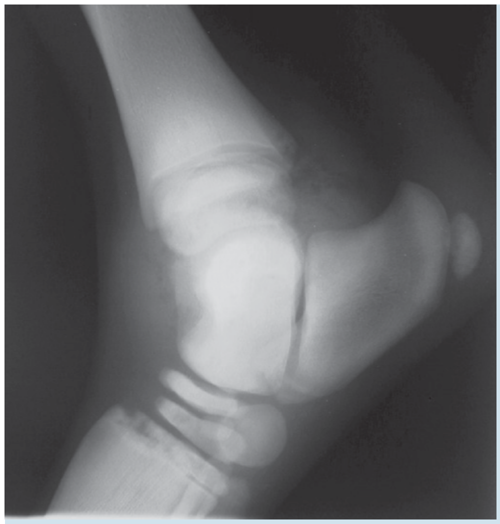


Figure 9.36: Grade 2 incomplete ossification of the tarsal joint.

Figure 9.37: Cranial-caudal radiograph of the carpus demonstrating a Grade 3 incomplete ossification of the carpal joint. Grade 3 is assigned when the cuboidal bones appear small and rounded with subsequent enlargement of joint spaces.

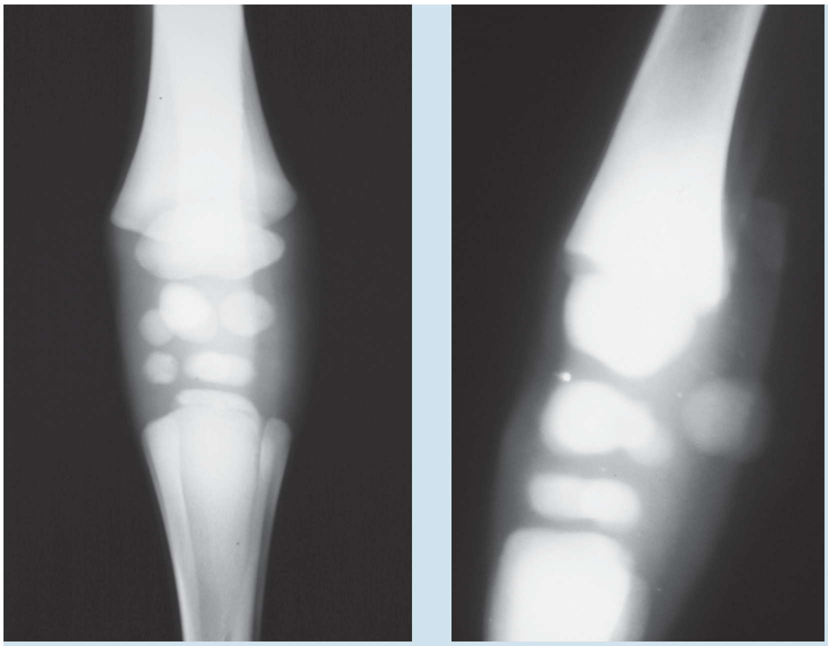


Figure 9.35a & b: Cranio-caudal and lateral radiographs of the carpus demonstrating Grade 2 incomplete ossification. Grade 2 is assigned when there is radiographic evidence of ossification of all of the cuboidal bones.

Figure 9.38: Cranio-caudal radiograph of the carpus demonstrating a Grade 4 incomplete ossification of the carpal joint. Radiograph from the foal in Fig 9.40. Grade 4 is assigned when the cuboidal bones are shaped like the adult counterparts and joint spaces are of expected width.

Figure 9.39: Foal whose radiographs are shown in Fig 9.35. This foal was born at 303 days of gestation. The mare had been treated for placentitis (diagnosed by ultrasonography) for 22 days prior to delivery. Note the sickle appearance to the hocks and flexor laxity. Although difficult to appreciate in this image there was a valgus deformity of both carpal joints.

Figure 9.41: Tarsal collapse. Note the "curby" appearance of the hock.

Figure 9.40: Same foal as Figs 9.35 and 9.39 at 60 days. Note the improvement in the carpal valgus and the improvement in the hock angle.

encouraged to stretch the tendons along with NSAIDs for analgesia. Surgery is not as successful and the need for surgery should be determined by carefully assessing the structures involved. Surgical management of fetlock contractures includes superior check ligament desmotomy (most commonly), inferior check ligament desmotomy (often combined with superior check ligament desmotomy), superficial digital flexor tenotomy (less commonly/severe cases), or transection of the suspensory (less commonly/severe cases).

Diagnosis

- Visual examination
- Digital examination may help distinguish tendon contracture from joint contracture
- Radiology is useful in making an initial diagnosis and serial radiographs are helpful in monitoring the progress of the condition
- Photographs are also useful for objectively monitoring progression and response to therapy.

Treatment

Medical management

- Treatment of acquired flexural deformities is aimed at correcting the primary problem for the contracture.
- Growth discrepancies due to excessive nutrition potentially respond to decreasing protein and energy content of the diet. A nutritionist should be consulted to formulate a suitable diet especially if there is more than one animal affected on the premises.

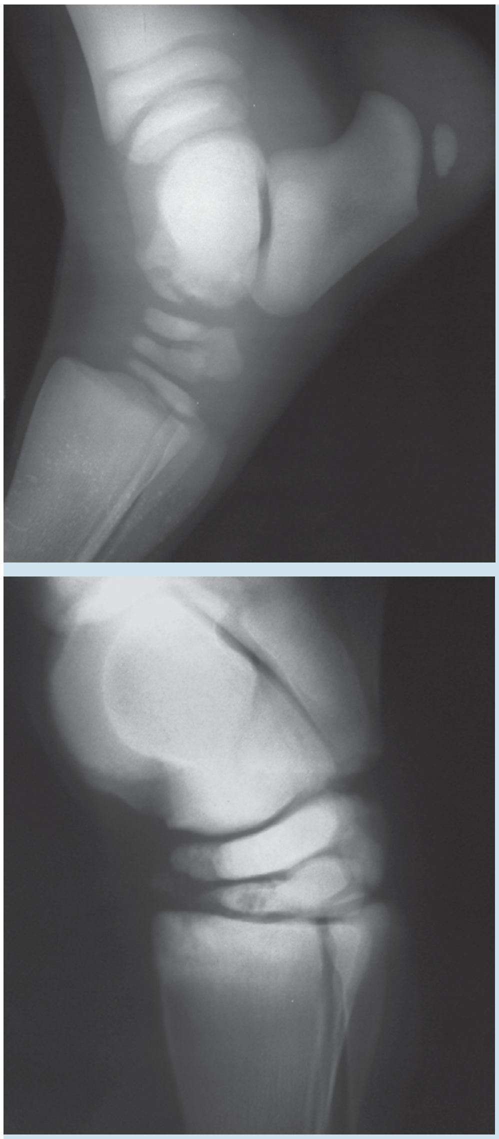


Figure 9.42: Grade 2 incomplete ossification of the tarsal bones was imaged in this foal at birth. A repeat radiographic examination 6 weeks later demonstrates crushing of both rows of tarsal bones.

Figure 9.43: Mild Type 1 club foot in a 1-month-old foal.

Figure 9.44: More severe but also Type 1 club foot in a 6-month-old foal. A glue-on shoe with toe extension has just been removed; some of the adhesive material can still be seen on the hoof wall. This foal had an inferior check ligament desmotomy performed 1 month prior to this image. Note also the distension of the other fetlock joint, associated with an OCD lesion.

- Controlled exercise programs and potentially exercise restriction is required in some cases.
- Low dose non-steroidal anti-inflammatory drugs (NSAIDs) can be used to help control mild levels of pain. Anti-ulcer medication should be used in conjunction with NSAIDs. Regular trimming/shoeing for distal interphalangeal contracture.
- Gradually lower the heel.
- Toe extensions.
- Bandaging/splinting/casting can be utilized to help provide soft tissue laxity.

Surgical management

- Distal interphalangeal joint contracture:
  - inferior check ligament desmotomy
  - deep digital flexor tenotomy

Figure 9.45: More severe (Type 2) club foot imaged here in an adult.

- Fetlock contracture:
  - superior check ligament desmotomy
- Carpal contracture:
  - ulnaris lateralis tenotomy.

Prognosis

- Mild cases – fair to good prognosis
- Severe cases – poor prognosis.

Angular limb deformities (Figs 9.46–9.58)

Angular limb deformities are very common conditions in all breeds. Angular limb deformity (ALD) refers to a lateral (outward) or medial (inward) deviation of a distal limb in a frontal plane. The terms valgus (outward deviation of the lower limb) and varus (inward deviation of the lower limb) are used to describe the type of deformity. Single or multiple deformities can occur and typically, the deviations described occur at the carpus, tarsus or the fetlock. Angular limb deformities can be congenital or acquired. If untreated, moderate to severe angular limb deformities can potentially lead to lameness and eventual osteoarthritis or degenerative joint disease.

The causes of angular limb deformities are generally multifactorial. Premature or dysmature neonates, which have incomplete ossification of the cuboidal bones and excessive laxity of peri-articular supporting structures, are often born with or develop angular limb deformities. Development of angular limb deformities in the postnatal foal is the result of the foal's body weight and a discrepancy in growth rate between the medial and lateral sides of the limb. Discrepancies in growth may occur anywhere along the bony column but most commonly involve the physis, epiphysis, or metaphysis. The etiology may be associated with focal physeal overloading, physeal trauma, or physeal inflammation. Factors associated with developmental orthopedic disease, such as mineral imbalances, other dietary or environmental factors, or genetics may also be involved.

Diagnosis

- Most angular limb deformities can be diagnosed from the clinical appearance of the limbs. The foals should be evaluated at rest but also at the walk to evaluate how the foal travels with each limb and how each foot contacts the ground surface.
- Radiographs can be taken to determine the degree of angulation, but it is very important to ensure the foal is standing squarely when taking the radiographs.

Treatment

- Management of angular limb deformities includes both conservative and surgical treatment options. Treatment options can vary for the particular age of the animal. In very young foals exhibiting signs of severe joint laxity or premature carpal/tarsal bones, strict stall rest is recommended until the laxity improves and the cuboidal bones have ossified.
- Conservative management includes restricting exercise, bandaging/splinting, nutritional changes, NSAIDs, and trimming the feet or application of hoof wall extensions. For valgus deviations, the outside hoof wall should be lowered slightly or an extension is

Figure 9.46: Bilateral carpal valgus angular deformity.

Figure 9.47: Carpal valgus angular deformity of the right forelimb.

Figure 9.48: Mild bilateral carpal valgus angular deformity of both forelimbs in a 12-hour-old foal. Conservative management of controlled exercise resulted in a normal appearance by 1 month of age.

Figure 9.49: (a) Metatarso-phalangeal varus angular deformity in a 1-day-old foal. This was managed with controlled exercise and lateral hoof wall extensions. (b) Same foal at 12 weeks of age.

Figure 9.50: Equilox™ lateral hoof wall extension. This has been placed to aid in the correction of a metacarpo-phalangeal varus.

Figure 9.51: Bilateral carpal and metacarpo-phalangeal varus angular deformity.

placed on the inside of the hoof wall. Medial hoof wall extensions encourage foals with carpal valgus to adduct the limb and turn in more when breaking over. This results in more physiologically normal strain on the limb, which should result in a straighter limb. For varus deviations, the inside hoof wall should be lowered slightly or an extension is placed on the outside of the hoof wall. Lowering the medial or lateral aspect of the hoof wall should result in an unbalanced hoof but this form of treatment has many apparent successes.

- Surgical management is based on two basic principles: growth acceleration or growth retardation. Growth acceleration is aimed at speeding the growth of the physis on the concave side of the deviation and growth retardation is aimed at slowing the growth of the physis on the convex side.
- For growth acceleration, hemicircumferential periosteal transection with periosteal elevation (HCPT/PE) is used commonly by many surgeons. This is a very simple surgery which can be performed in the field and multiple times if necessary. This technique is typically used for the younger foals to try and coordinate the surgery with a potential growth stage of the foal. This procedure is

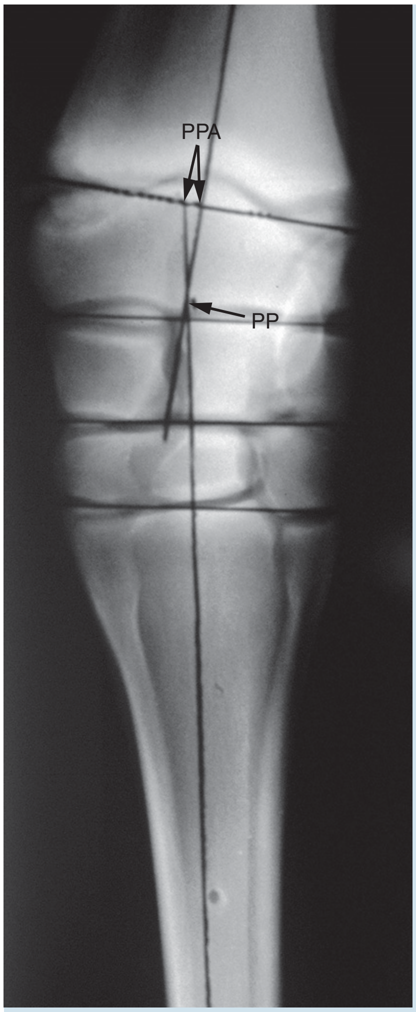


Figure 9.52: Dorso-palmar radiographic view of a carpus with valgus deformity. Medial is to the left. Lines are drawn parallel to the long axis of the radius and third metacarpal bone. The point where these lines intersect is known as the pivot point (PP), and the angle between them is the pivot point angle (PPA).

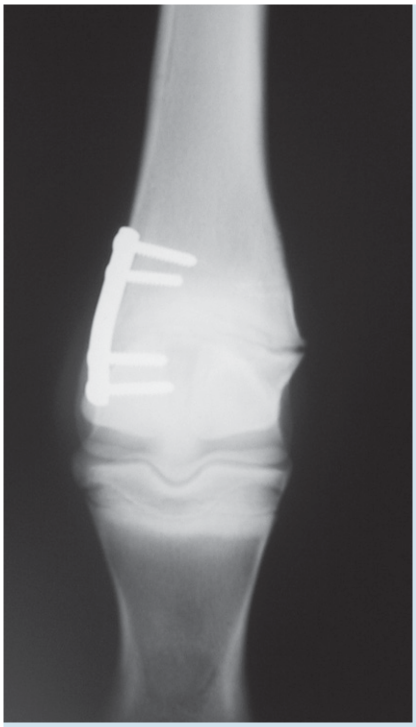


Figure 9.54: Metacarpo-phalangeal radiograph demonstrating transphyseal bridging with a "plate and screw" technique.

Figure 9.53: Carpal radiographs demonstrating transphyseal bridging with the screw and wire technique.

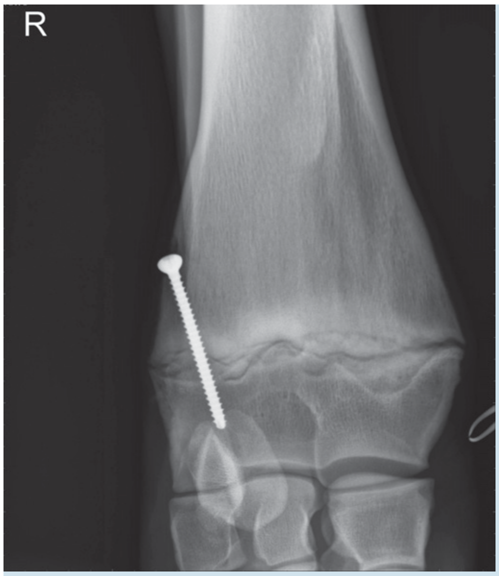


Figure 9.55: Carpal radiograph demonstrating transphyseal bridging using a "single screw" technique.

Figure 9.56: Carpal radiograph demonstrating transphyseal bridging using a "staple."

Figure 9.57: Placement of a transphyseal staple. Before the incision is made, the skin over the distal medial radius is rolled cranially 2 cm, and the physis is identified with a needle. With a #15 scalpel blade, a 3 cm proximal to distal incision centered over the physis is made through the skin and subcutaneous tissues to the level of the periosteum. This incision should be 1.5–2 cm caudal to the medial prominence of the radial physis. The transphyseal staple is then centered on the incision and is driven to the level of the skin with a mallet. The skin is sutured and a pressure bandage is applied. The foal should remain confined to a box for 1–2 weeks.

commonly performed around 2–4 weeks of age. The surgical technique of HCPT/PE may be performed medially or laterally (depending on the deformity present) at the distal radius, distal metacarpus/metatarsus, or distal tibia.

- For growth retardation, transphyseal bridging (TPB) techniques are commonly used by many surgeons. Transphyseal bridging may be performed with staples, screws and wires, screws and a plate, or the single screw technique. The surgeon's preference usually dictates the method chosen, but the single screw technique and screws and wires are currently the most common methods used for transphyseal bridging. All techniques are easy to perform and all require a second surgery to remove the implants. The implants should be removed once the limb has corrected to the appropriate angulation. If the implants are not removed the limb can potentially overcorrect. The surgical technique of TPB may be performed medially or laterally (depending on the deformity present) at the distal radius, distal metacarpus/metatarsus, or distal tibia.
- Once the growth plates have closed or calcified, none of the above techniques will correct an angular limb deformity. Trimming and shoeing can help mild deviations. Corrective wedge osteotomy/ostectomy can be used to potentially correct moderate to severe deviations. This is an expensive and involved procedure, and often times will not provide an athletically sound animal.

Figure 9.58: Removal of a transphyseal staple. Removal of a transphyseal staple should occur as soon as the leg has straightened. After the skin is rolled cranially the staple is located with a needle. The skin and subcutaneous tissues are sharply dissected to the level of the staple with a #15 scalpel blade, which is also used to dissect over the cranial and caudal edges of the staple. An elevator is placed between the bone and the body of the staple, which is then levered free. Closure, bandaging and aftercare are similar to those following staple placement.

Hemicircumferential periosteal transaction / periosteal elevation (Fig 9.59)

Surgical technique:

- The procedure is typically performed over the most medial or lateral aspect of the physis involved.
- The location of the skin incision is about 10 mm proximal to the physis.
- Using a #15 scalpel blade, a small 10 mm longitudinal incision is made through the skin. The incision is then extended into the deeper fascia and periosteum.
- The longitudinal incision in the periosteum should be about 20–30 mm long, starting about 10–20 mm proximal to the physis.
- The blade is then positioned perpendicular to the distal end of the periosteal incision. A second incision is then made through the periosteum to make an inverted "T."
- A periosteal elevator is introduced through the skin incision and placed into the vertical periosteal incision. The cranial and caudal triangular periosteal sections are elevated from the underlying bone.
- In young foals the rudimentary ulna is transected with the periosteum using a scalpel blade. In older foals or foals with an ossified ulna, a rongeur may be required for transection.
- The skin incision is not closed. A bandage is placed over the incision site for 2–3 days.
- The foal is confined to a stall for 1 day, and then allowed paddock or pasture turnout.
- Postoperative antimicrobials are given for 3–5 days, and the bandage is removed in 5–7 days.
- Tetanus prophylaxis is given if indicated (if the mare was not vaccinated in the 2–4 week period prior to foaling).

Septic arthritis (Figs 9.60–9.107)

Equine orthopedic infections are commonly observed in the foal following bacteremia or septicemia, lacerations, or can occur idiopathically. The establishment of an infection is determined by the host's defense, virulence of the organism, and local factors. In foals, bacteremia or septicemia originating from the umbilicus, respiratory tract, or intestinal tract can cause orthopedic infections just after birth or as late as 30–45 days after birth. This period of vulnerability is often dependent on the level of maternal protection. Failure of passive transfer (FPT) is the highest risk factor for development of septicemia in foals. In one study, the risk of potential disease as a result of FPT is reported to be as high as 78%.

Joint sepsis in the foal has been classified into four general types: E, S, P, and T. This classification scheme is based on categorizing the infections based on location.

- *Type S* (synovial), where a septic arthritis resulting from inoculation of the synovial membrane is present.
- *Type E* (epiphysis), where subchondral bone infection is present.
- *Type P* (physis), where infection of the physis on the metaphyseal side of the growth plate is identified.
- *Type T* (tarsal/cuboidal), where the infection involves the tarsal and carpal cuboidal bones.

The most common types observed by the author are S and E, but the others do occur commonly.

Clinical signs and diagnosis

- Orthopedic infections are often very obvious to identify, but in some cases the exact site can be difficult to determine. The classical clinical signs of joint infections are ***lameness, fever*** and ***joint distension. Any lame neonatal foal should be regarded as having septic osteoarthritis until proven otherwise.*** As such any lame neonatal foal should be placed on broad-spectrum antibiotics until all diagnostic tests have been completed.
- Joint sepsis involving the distal limbs often results in appreciable joint effusion due to the increased vascular permeability of the joint capsule. However, joint effusion of the proximal limbs can be hard to appreciate visually, but digital palpation may elicit a painful response.
- In foals, osteomyelitis due to septicemia/bacteremia can potentially be challenging to identify when the involvement is in the pelvis and vertebral bodies. Sepsis can also develop in other synovial structures such as tendon sheaths and bursas.
- Standard diagnostic tests include complete blood counts and fibrinogen, and radiographs.
- In some cases ultrasonography, nuclear scintigraphy, computed tomography (CT), and magnetic resonance imaging (MRI) may be used to help identify the source of infection.

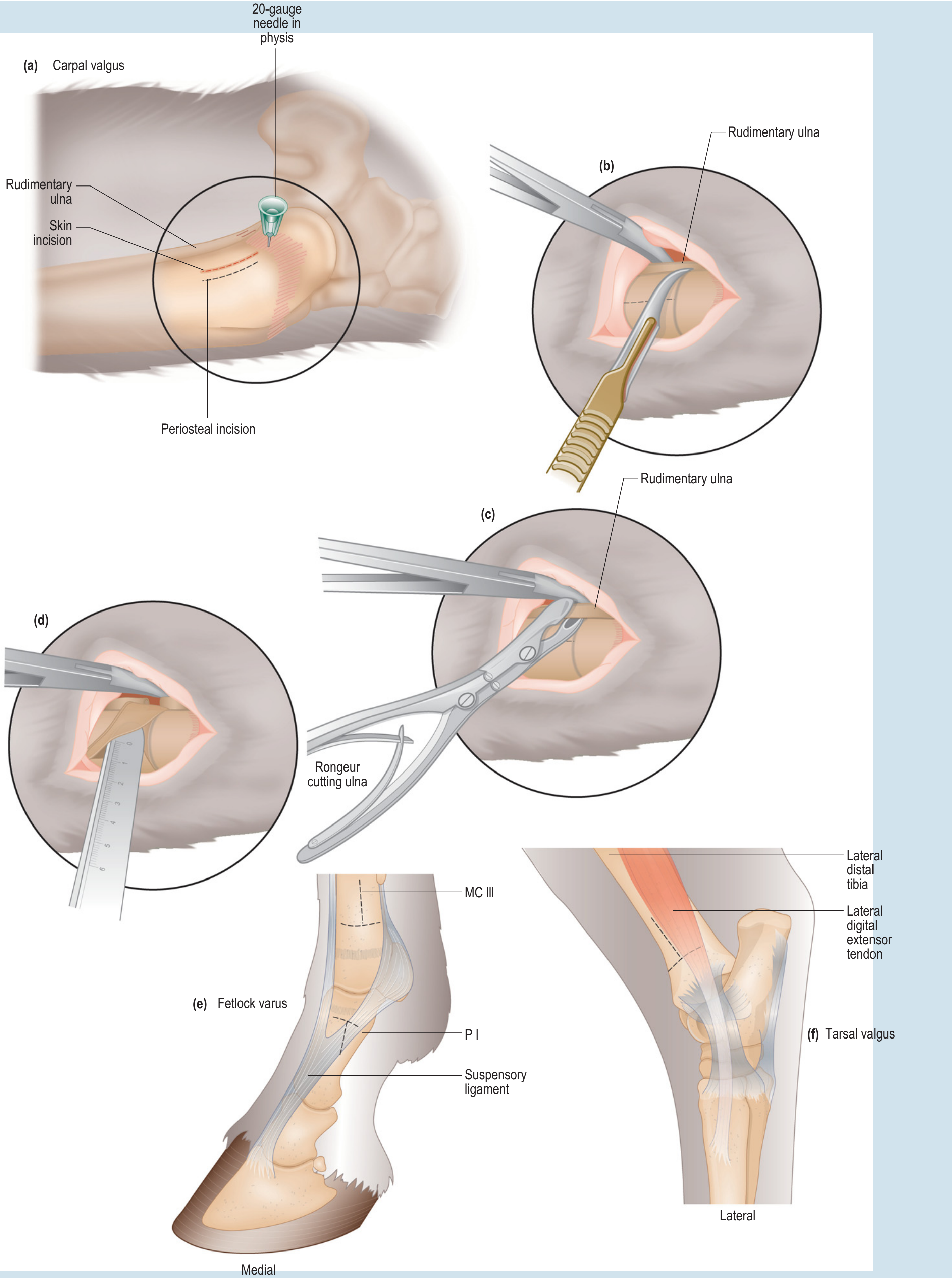


Figure 9.59: (a–f) Hemicircumferential periosteal transaction/periosteal elevation for correction of carpal valgus (a–d), fetlock varus (e) and tarsal valgus (f). A full description of the technique is given in the text. (From Adams SB, Fessler JF 2000 Atlas of equine surgery. Saunders, Philadelphia, p 365.)

Figure 9.60: Swelling of the distal limb in a 1-month-old foal was accompanied by a non-weight bearing lameness. Clipping the limb and a closer digital examination revealed effusion of the distal interphalangeal joint which is evident as a soft palpable swelling at the coronary band.

Figure 9.61: Postmortem specimen from the foal in Fig 9.60 demonstrating extensive lysis of the extensor process of P3.

Figure 9.62: Marked swelling of the metacarpo-phalangeal joints in an Arabian foal with osteoarthritis.

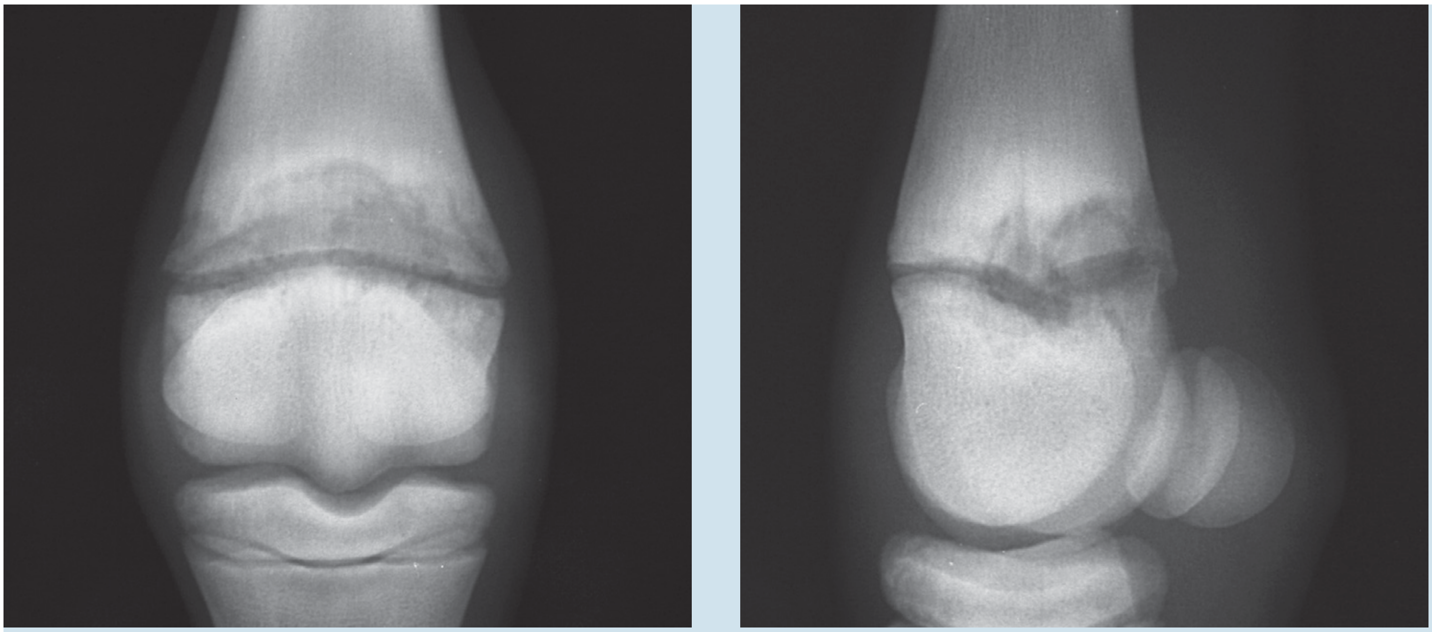


Figure 9.63: Dorso-palmar and lateral radiographs of the **left** metacarpo-phalangeal joint from the foal in Fig 9.62, demonstrating Type P osteomyelitis. Note the extensive lysis along the physis.

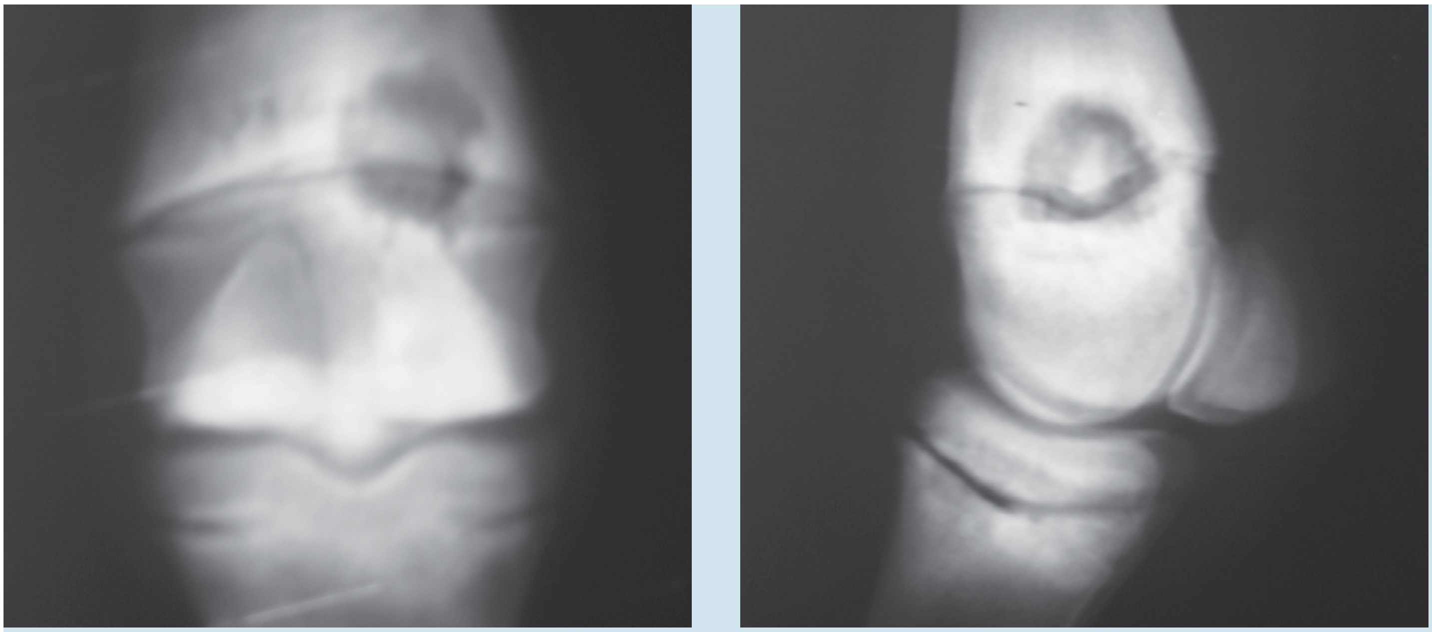


Figure 9.64: Dorso-palmar and lateral radiographs of the **right** metacarpo-phalangeal joint from the foal in Fig 9.62. Note the well defined sequestrum.

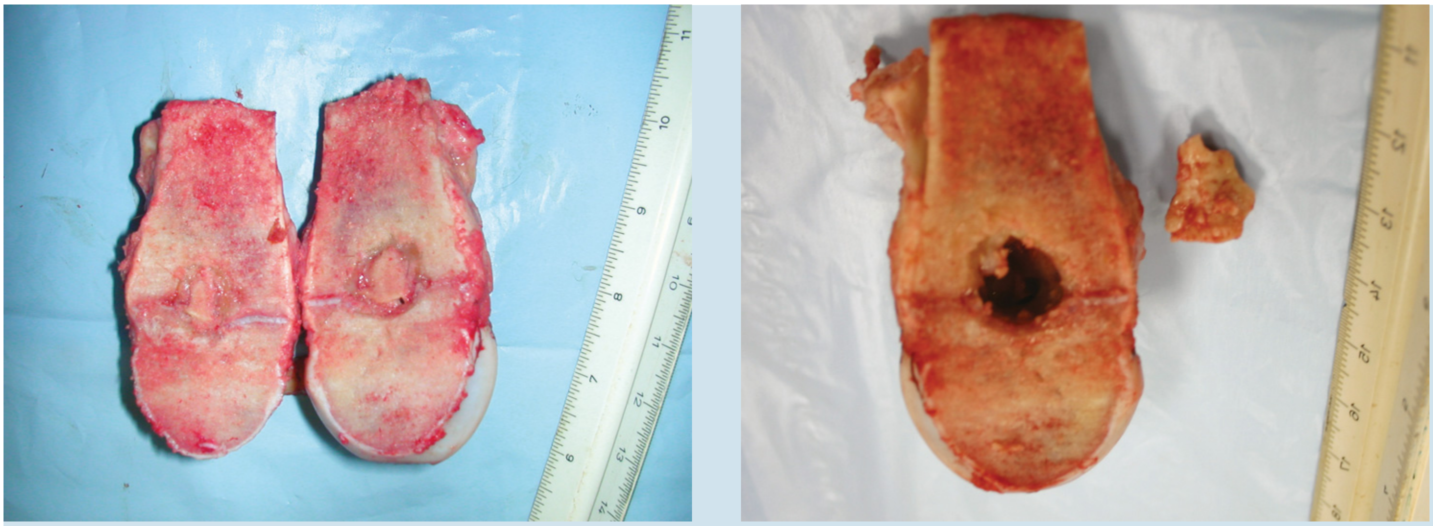


Figure 9.65: Postmortem specimen from the foal in Figs 9.62 and 9.64; note the necrotic sequestrum.

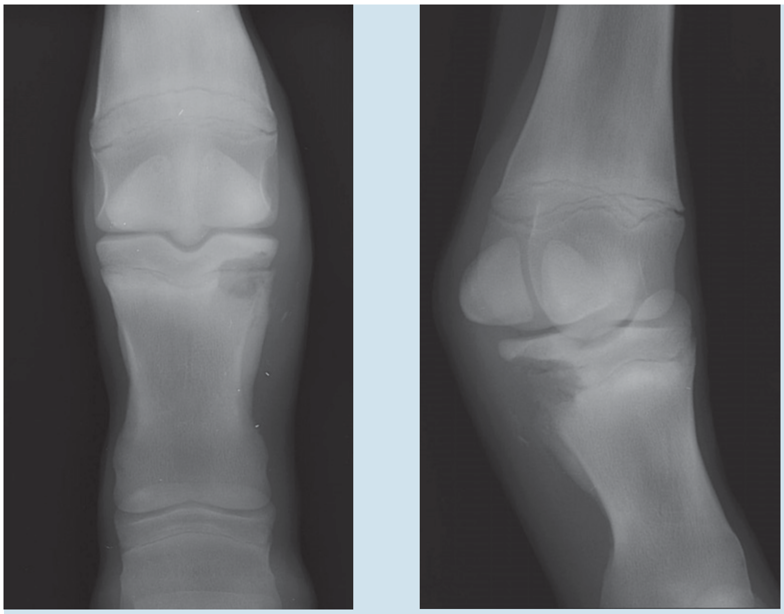


Figure 9.66: Dorso-palmar and dorsolateral–palmarmedial radiographs of the left fetlock joint of a foal demonstrating a large lytic lesion of the proximal physis of the first phalangeal bone (Type P septic osteoarthritis).

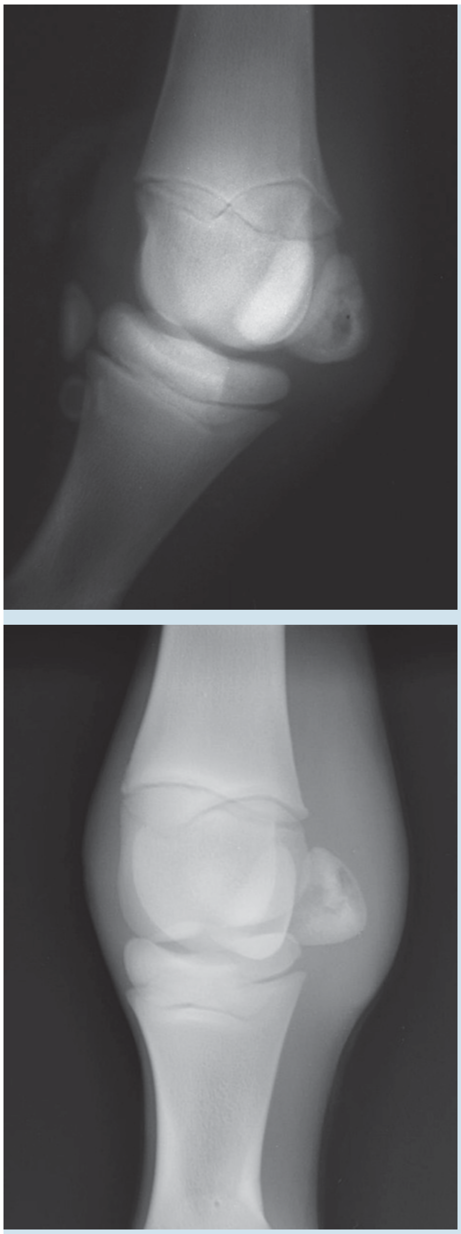
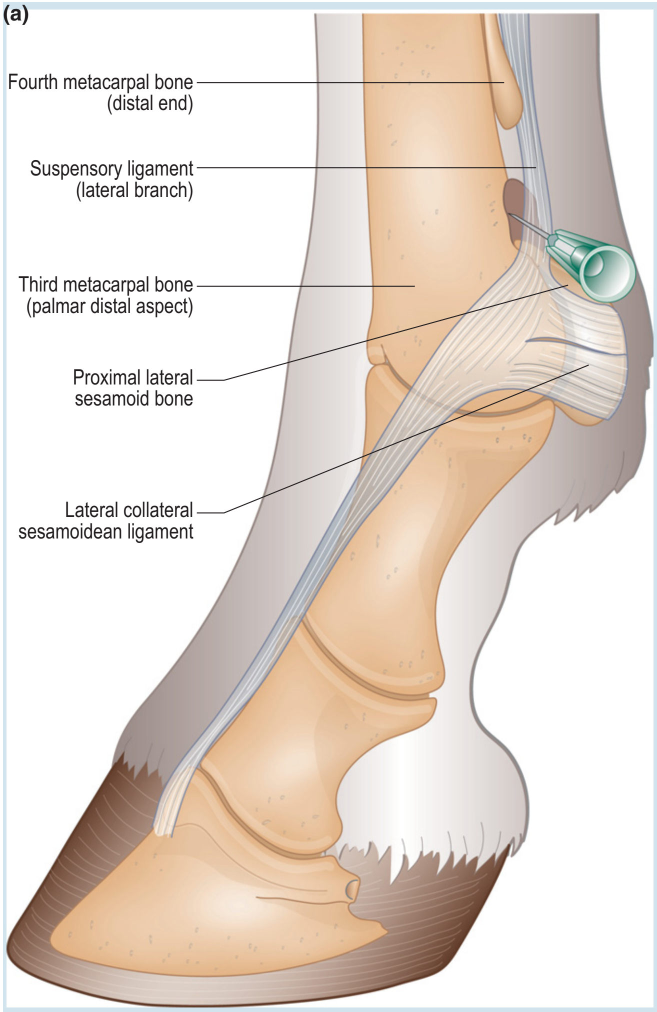


Figure 9.67: Radiographs of fetlock joints from two different foals demonstrating Type T osteoarthritis of a single sesamoid in each joint.

(a)



(b)

Figure 9.68: Arthrocentesis of the fetlock joint. The joint can be approached through the lateral aspect of the palmar or plantar pouch (illustrated), which is located between the following palpable structures:

– The palmarodistal/plantarodistal aspect of the cannon bone.
– The dorsal edge of the lateral branch of the suspensory ligament.
– The distal end of the lateral splint bone.
– The proximal aspect of the lateral sesamoid bone.

The needle is directed slightly downward.

(b) The fetlock joint can also be entered cranially on either side of the extensor tendon. The fetlock joint is readily entered at this site when there is marked effusion of the joint. The advantage of this site is that inadvertent penetration of the metacarpal or metatarsal blood vessels is less likely.

Figure 9.69: Radiograph demonstrating septic osteomyelitis of the third metatarsal bone (cannon) in a 4-month-old foal. This foal had previously been diagnosed with salmonellosis. The foal received a prolonged course of antibiotic therapy in conjunction with hyperbaric oxygen therapy.

Figure 9.70: Radiograph of the same foal as Fig 9.69 3 weeks later, showing a reduction in the size of the osteomyelitis lesion. Note the marked sclerotic rim associated with the lesion at this point.

Figure 9.71: Lateral radiograph of the same foal as Figs 9.69 and 9.70 5 weeks later. There is considerable improvement with a marked decrease in the size of the lytic lesion. The foal was sound at this point.

Figure 9.72: Marked swelling of the carpus in a foal with osteoarthritis.

Figure 9.73: Radiograph demonstrating Type P osteoarthritis of the radius. Note the well defined lytic area.

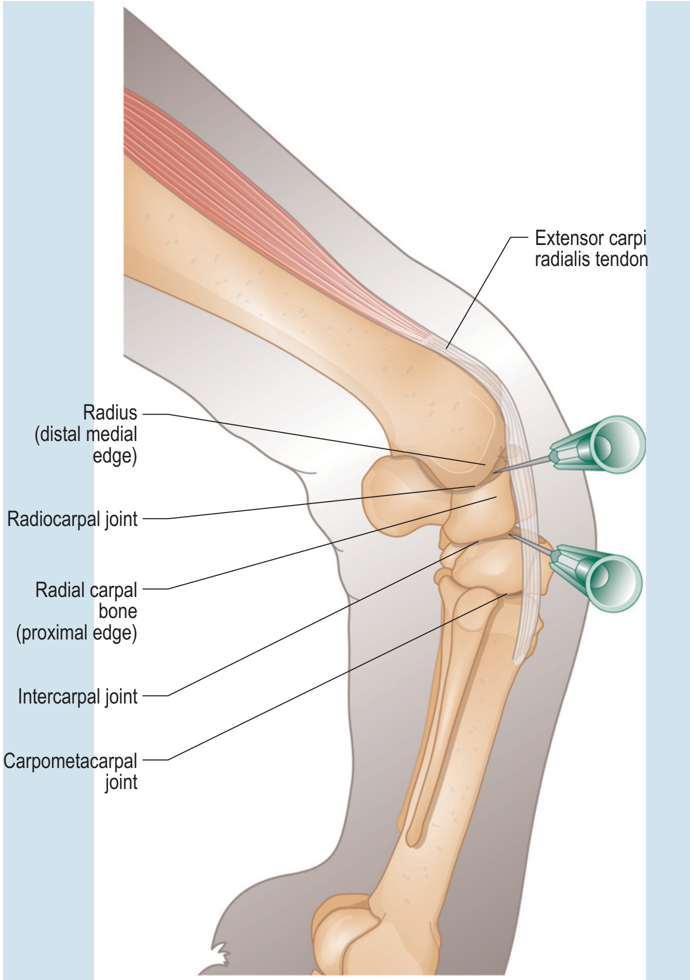


Figure 9.74: Technique for arthrocentesis of the carpal joint using a dorsal approach (illustrated). The limb is flexed and the radiocarpal joint is located by palpating the medial aspect of the distal edge of the radius and the proximal edge of the radial carpal bone. The needle is inserted midway between these two structures.

- The intercarpal joint is located by palpating the distal edge of the radial carpal bone and the medial aspect of the proximal edge of the third carpal bone with the limb in a flexed position. The carpometacarpal joint communicates with the intercarpal joint and does not normally require separate entry.
- The radiocarpal and intercarpal joints can also be entered laterally. The tendons of the ulnaris lateralis and lateral digital extensor muscles converge to form a "V" on the palmarolateral aspect of the limb. A small depression can be palpated distal to the "V" and this is the site for needle insertion for arthrocentesis of the radiocarpal joint.
- The intercarpal joint is entered approximately 1 inch distal to the point of insertion for the radiocarpal joint. A 20G needle is sufficient for arthrocentesis. An 18G needle may be used for through and through lavage.

Figure 9.75: Through and through joint lavage performed on the intercarpal joint. The ingress needle is placed in the palmar aspect and two egress needles are placed in the dorsal aspect of the intercarpal joint.

Figure 9.76: Marked swelling of the right tarsal joint in a foal with septic osteoarthritis. Joint swelling and marked lameness are usually key features of septic osteoarthritis.

Figure 9.77: Swelling of the left tarsal joint as a result of trauma to the lateral aspect of the joint resulting in hematoma formation. This foal was not lame and digital palpation revealed that there was no effusion in the tibiotarsal joint and that the swelling was limited to the soft tissue of the tarsus.

Figure 9.78: Severe swelling of the left tarsal joint in a 4-month-old foal. This foal was known to have an osteochondrosis lesion on the medial malleolus with associated joint effusion and was stabled with another foal. The swelling and degree of lameness were noted to have increased dramatically in the 24 hours prior to this photograph. Radiographs revealed no additional abnormalities. CBC and fibrinogen measurements were within normal limits, as was rectal temperature. A septic osteoarthritis was considered to be unlikely given the age of the foal and normal blood parameters. Ultrasonography of the joint was then performed which revealed severe disruption of the lateral collateral ligaments.

Figure 9.79: Dorso-plantar radiograph of the foal in Fig 9.78, 6 months later showing marked bony proliferation of all the bones on the lateral aspect of the joint (arrows). This foal was euthanized due to the poor prognosis for athletic function.

Table 9.1. Synovial fluid cytology for various clinical conditions

Parameter	Normal	Mild synovitis (e.g. OCD)	Osteoarthritis	Infectious arthritis
Total Leukocytes (per μL)	50–500	20–250	$\leq 1 \times 10^3$	$20–200 \times 10^3$
Neutrophils (%)	<10	<10	<15	>90 (variable toxic changes)
Mononuclear cells (%)	>90	>90	>85	<10
Total protein (g/dL)	0.8–2.5	0.8–3.0	0.8–3.5	4.0–8.0 +

Figure 9.80: Swelling of the right tarsus in a 3-week-old foal.

Synovial fluid cytology (Table 9.1) can not only be used for diagnosis of infectious arthritis but also can be useful for differentiating infectious processes from other conditions.

Treatment

- Treatment of orthopedic infections in foals is aimed at controlling and efficiently eliminating the infection.
- *Systemic antibiotics, anti-ulcer medication, and anti-inflammatory agents are used in all cases.*
- The type of antibiotic chosen depends on the organism involved (or suspected) and sensitivity results, in addition to the stage of disease and surgeon's preferences.
- Common antibiotics used initially include ampicillin, sodium or potassium penicillin, oxytetracycline, gentamicin, amikacin, ceftiofur and cefazolin.
- In select cases a third generation cephalosporin (ceftazidime/cefotaxime) or an imipenem/cilastatin combination can be used. Vancomycin can be used for methicillin resistant *Staphylococcus aureus* (MRSA) infections. However, these should be used only if antibiotic resistance patterns require the use of such antibiotics.
- ***Fluroquinolone antibiotics (e.g. enrofloxacin and marbofloxacin) should be used with extreme caution.*** Enrofloxacin is not licensed for use in juvenile animals due to its potentially deleterious effects on cartilage. The use of enrofloxacin may be associated with joint effusion or tendon laxity. If either of these is seen, use of the drug should be discontinued immediately. In cases of septic osteoarthritis it may be difficult to discern if joint effusion has worsened. Continued use of the drug after signs of joint inflammation have become apparent can result in permanent cartilaginous damage and lameness. ***If it is necessary to use this drug on the***

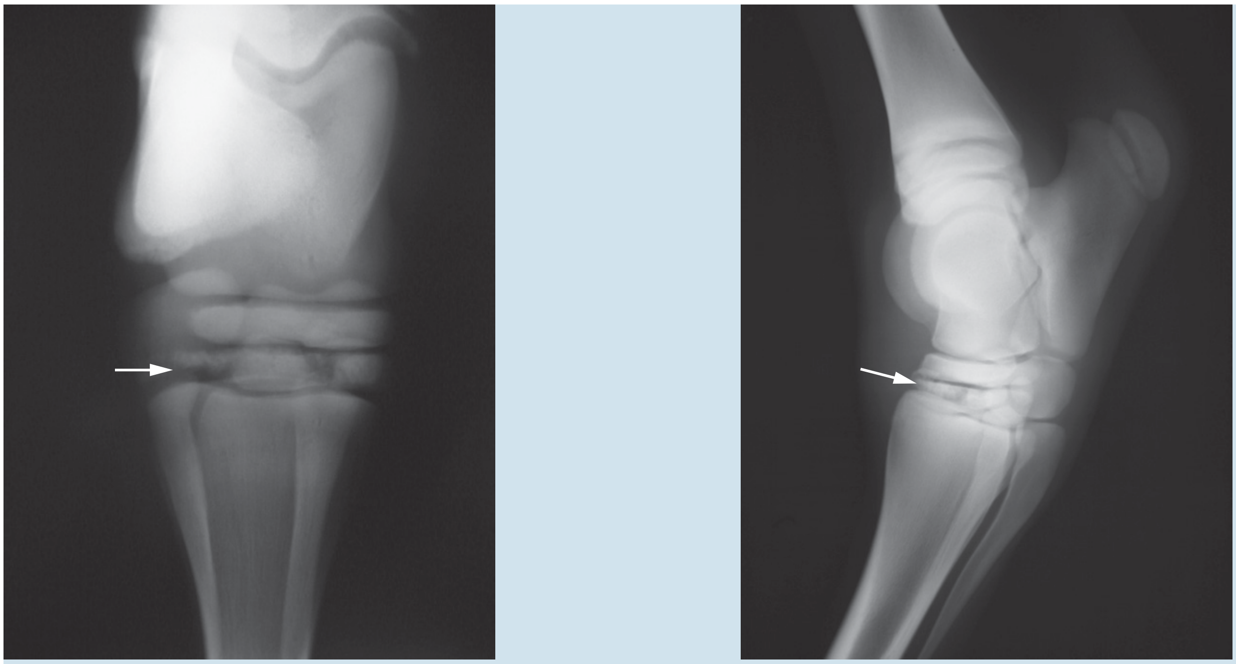


Figure 9.81: Dorso-plantar and lateral radiographs of the right tarsus of the foal in Fig 9.80, demonstrating Type T septic osteoarthritis. Note the lysis in the distal row of tarsal bones (arrows).

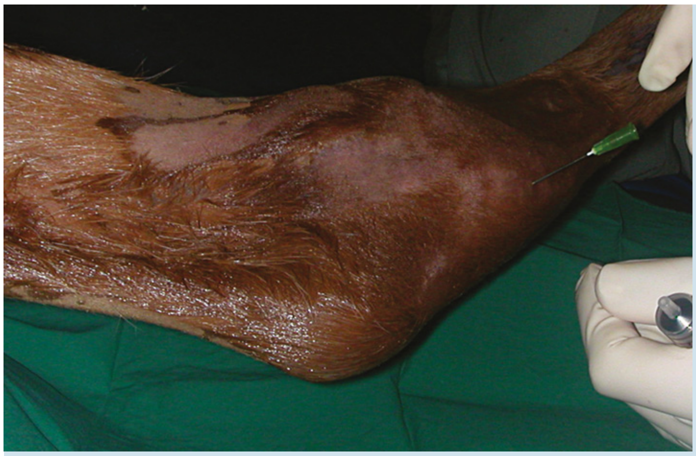


Figure 9.82: Intraosseous injection of the distal row of tarsal bones in the same foal as in Figs 9.80 and 9.81. This treatment was combined with systemic antibiotics, antibiotic injection of the distal inter-tarsal joint (Fig 9.83) and regional antibiotic perfusion (Fig 9.84).

Figure 9.83: Injection of the distal inter-tarsal joint of the foal in Figs 9.80 & 9.81.

Figure 9.84: Regional antibiotic perfusion of the tarsus. Same foal as Figs 9.80–9.83.

Figure 9.85: Lateral radiograph of the same foal as Figs 9.80–9.84 2 weeks later. Note the lysis is more extensive and there is crushing of the distal row of metatarsal bones. This foal also had severe osteoarthritis of the elbow (Figs 9.94–9.96) and was euthanized.

basis of sensitivity testing it should be combined with chondroprotective agents.

- In cases of synovial infections, through and through lavage of the synovial structure is commonly performed. In performing a synovial lavage it is extremely important to collect a sample for cytology and one for culture and sensitivity. The procedure can be performed standing or under general anesthesia (easier).
  - The affected joint is clipped and aseptically prepped
  - Try to insert the first needle (14–16G) in the most distended portion of joint
  - Collect a sample for culture/sensitivity and cytological analysis either by aspiration or free flow of synovial fluid
  - Attach a fluid line and distend the joint with a small volume of the solution to be used
  - Try to insert the second needle the farthest distance possible from the first needle
  - Lavage joint with 500 mL to 1000 mL of sterile balanced electrolyte solution ± antibiotics (gentamicin or amikacin)
  - Infuse 3–5 mL of amikacin into the joint
  - The limb is then bandaged if the affected joint is below the tibia or radius
- Regional limb perfusion (either intravenous or intraosseous) can be performed in select cases which do not respond to systemic antibiotics and require high concentrations of antibiotics locally (Table 9.2).
  - A tourniquet is secured proximal to the infected site (depending on location two tourniquets may be needed; one applied above and one below the infected focus). Tourniquet period is 25–30 minutes. The use of wide tourniquets (20% wider than the limb diameter) decreases the chances of trauma to the underlying tissue.
  - For intravenous regional limb perfusion a superficial vein is catheterized whereas for intraosseous regional limb perfusion the solution is directly injected into the medullary cavity of bone.
  - The intra-arterial route has been used for perfusions in the past but drug-induced toxic effects on the endothelium can be more severe in the arteries than in the veins, so the arterial route is now discouraged in clinical practice.
  - Either an Esmarch bandage or surgical rubber tubing can be used to occlude the arterial and venous systems therefore preventing the blood from refilling the vasculature and the perfusate from leaking.
  - Culture and susceptibility tests should be used to help guide antimicrobial selection.
  - The optimal antimicrobial dose or volume of perfusate is not accurately known. Some authors recommend administration of 1/3 of the daily systemic dose as a general rule for distal limb perfusion.
  - When antimicrobials with known systemic toxic effects (e.g. aminoglycosides) are used, concurrent systemic administration of the antimicrobial should be delayed or plasma drug concentrations monitored to avoid exceeding the toxicity threshold because after releasing the tourniquet, plasma concentrations *may* increase substantially.
  - Regional limb perfusion is usually performed once daily to once every 2–3 days depending on the severity of the case.
- In cases requiring more aggressive treatment, an arthrotomy may be required to help eliminate fibrin from the joint.
- In severe cases involving sites of osteomyelitis, removal of bone sequestrums or necrotic bone may be required.

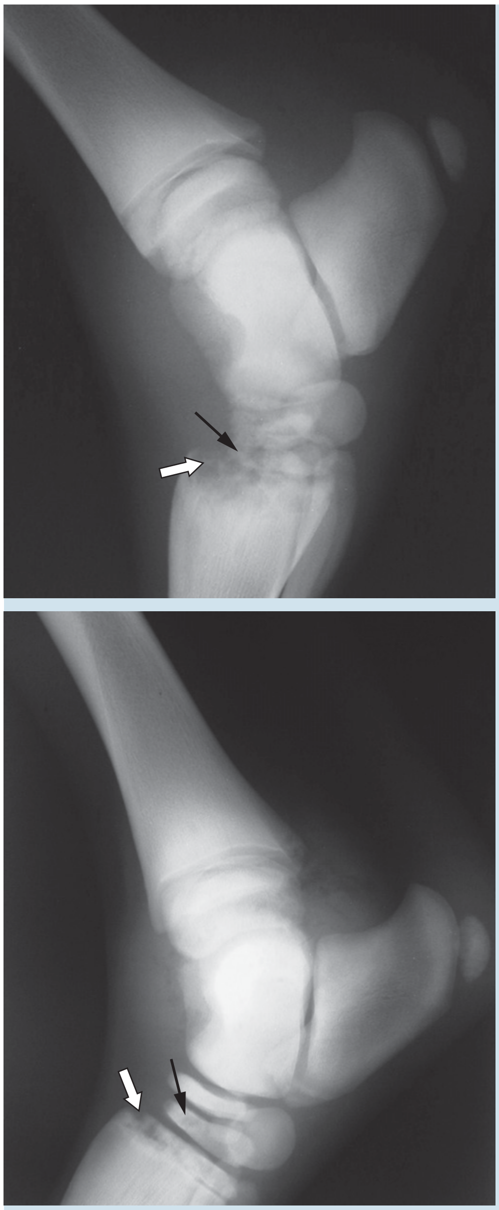


Figure 9.86: Lateral radiographs of the tarsus demonstrating Type E and Type T septic osteoarthritis. Note the lytic areas in the proximal third metatarsal bone (light arrows) in addition to the lytic areas in the tarsal bones (dark arrows).

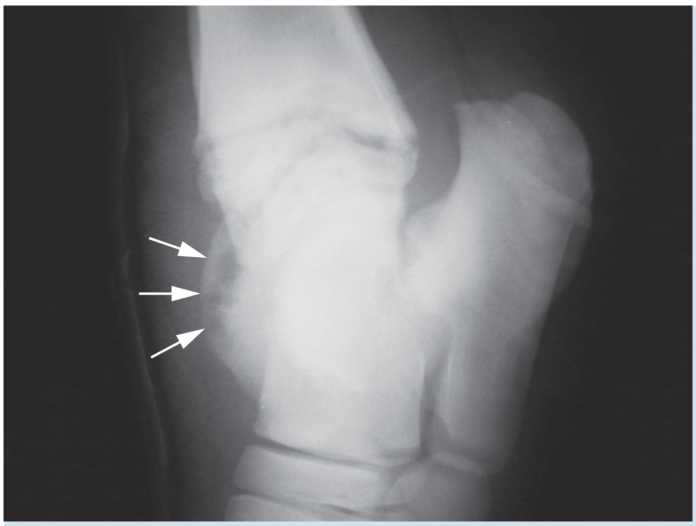


Figure 9.87: Lateral radiograph of the tarsus demonstrating a lytic osteoarthritis lesion of the lateral trochlear ridge of the talus (arrows).

Figure 9.88: Lateral radiograph of the hock demonstrating severe osteomyelitis of the apophysis of the calcaneus. This foal was enthanized due to the severity of the lesion.

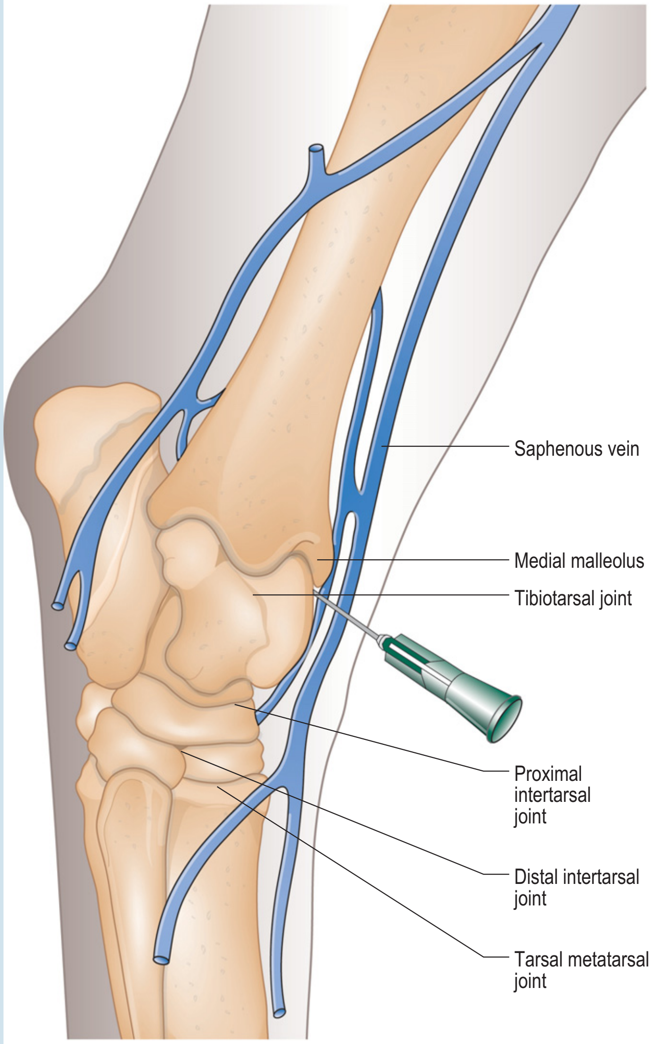


Figure 9.89: The hock is composed of four joints: the tibiotarsal, proximal intertarsal, distal intertarsal and tarsometatarsal joints. The tibiotarsal and proximal intertarsal joints communicate directly. There is individual variation as to the communication of the distal intertarsal and tarsometatarsal joints. Technique for arthrocentesis of the hock (tibiotarsal joint) is illustrated here. This joint is penetrated on either side of the saphenous vein as it vertically traverses the joint, approximately 1–1 ½ inches distal to the level of the prominent medial malleolus of the tibia. The distal intertarsal joint is entered on the distomedial aspect of the joint, using a 1 inch 20–23-gauge needle just below the palpable distal edge of the cunean tendon into a small T-shaped gap formed by the junction of the fused first and second tarsal bones, the third tarsal bone and the central tarsal bone.

Figure 9.90: Through and through lavage of the tibio-tarsal joint. Needles have been placed in the dorsal medial, dorsal lateral and plantar lateral aspects of the joint.

Figure 9.92: Marked effusion of the elbow joint. An 18G 1-inch needle is being placed in the joint to aspirate a sample for culture and cytology. This joint was then flushed through and through and antibiotics were infused into the joint.

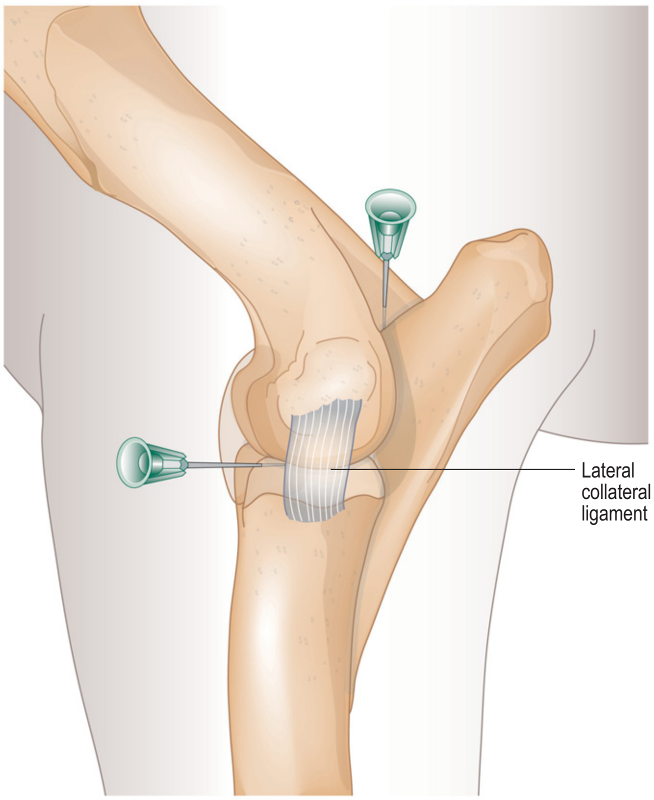


Figure 9.91: The elbow joint is the combined humeroradial, humeroulnar and radioulnar joints. Technique for lateral arthrocentesis of the elbow joint involves entering the joint using a 1 ½ inch needle (1 inch may suffice in very young foals) cranial or caudal to the palpable collateral ligament, two-thirds of the distance between the lateral humeral epicondyle and the radial tuberosity. The large caudal pouch of the joint can also be entered through the olecranon fossa but involves passing through the triceps muscle (from Hardy J, Latimer F 2003 Orthopedic disorders in the neonatal foal. Clinical Techniques in Equine Practice 2:96–119).

Figure 9.93: Aspiration of synovial fluid from an elbow joint with septic osteoarthritis. Note the bright yellow color of the fluid. Normal synovial fluid has a clear to light straw color appearance. Under normal conditions it is difficult to aspirate fluid from this joint.

Figure 9.94: Radiograph of a humeroradial joint with Type P osteomyelitis. Note the large circular area of lysis adjacent to the distal humeral physis (arrows).

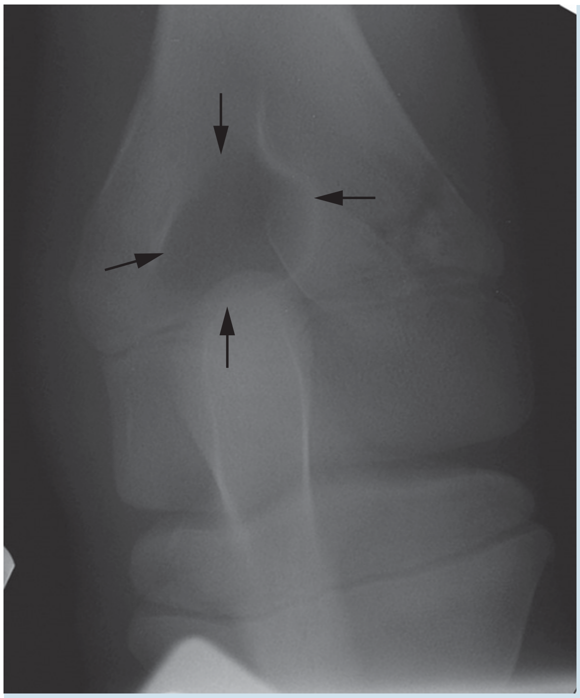


Figure 9.95: Dorso-palmar radiograph of the humeroradial joint demonstrating osteomyelitis of the distal humerus. Note the circular lytic area (arrows).

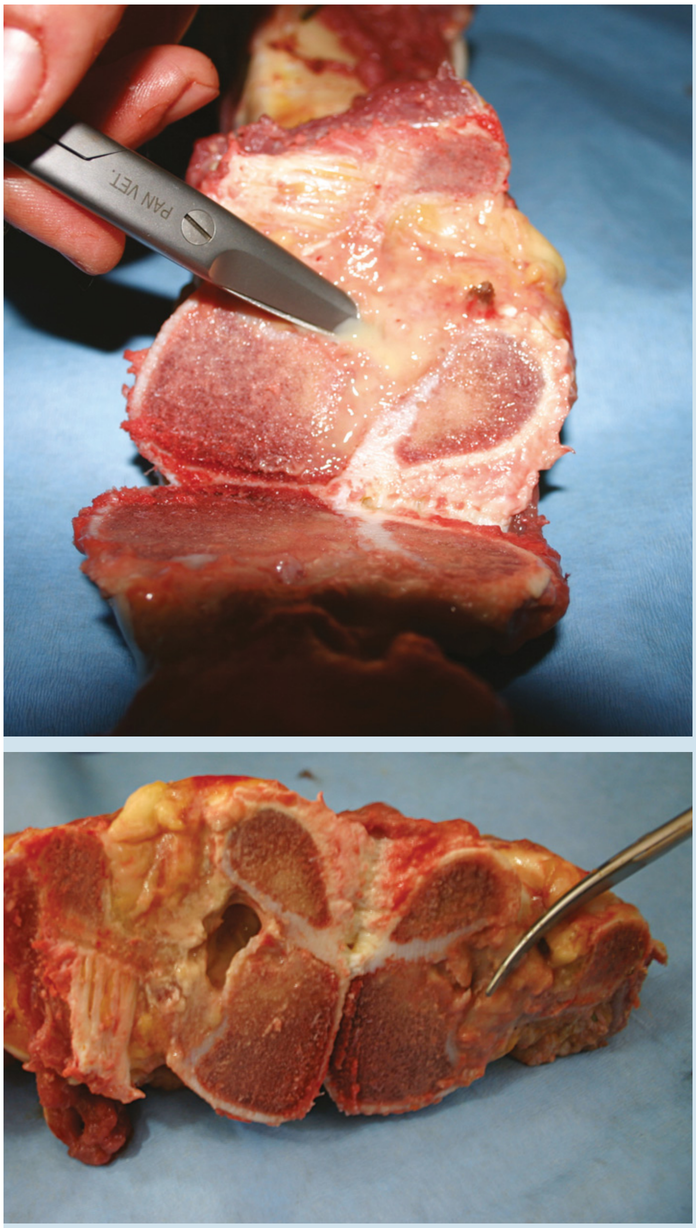


Figure 9.96: Postmortem specimen from the foal in Fig 9.94. Bone lysis has resulted in a large cavitary lesion which is full of purulent material.

Figure 9.97: Through and through lavage of the elbow joint.

Figure 9.98: Effusion of the patello-tibial joint in a foal with Type S osteoarthritis. This foal had no apparent radiographic changes but a synovial fluid aspirate indicated an elevation in WCC and protein.

Figure 9.99: Marked swelling of the stifle area in a foal with septic osteoarthritis that had previously had enterocolitis.

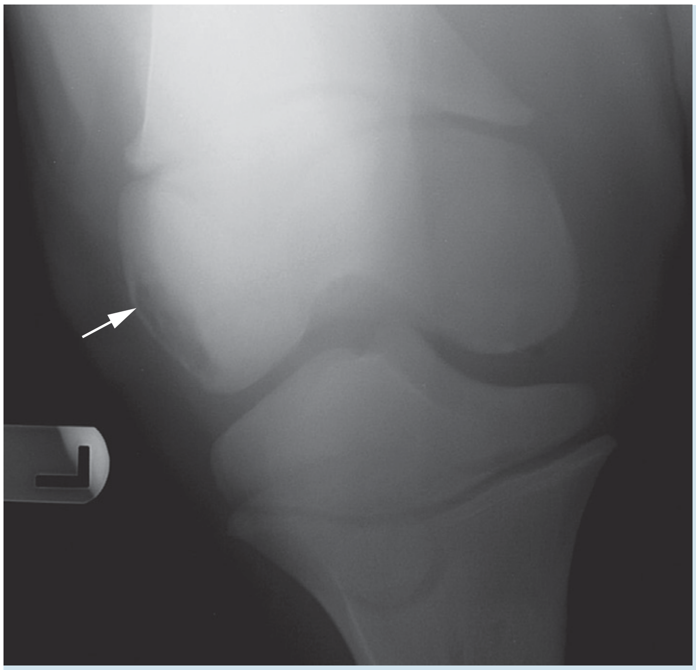


Figure 9.100: Caudo-cranial radiograph of the stifle demonstrating Type E osteoarthritis of the lateral condyle of the femur (arrow)

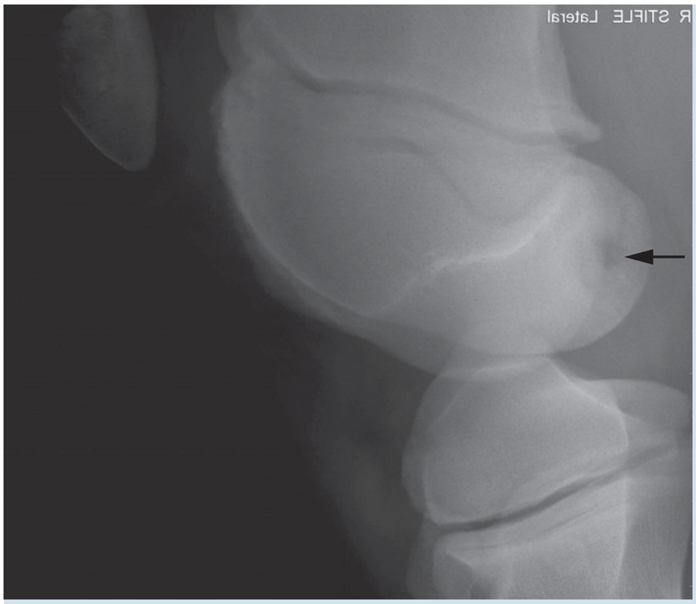


Figure 9.101: Lateral radiograph of the stifle demonstrating Type E osteoarthritis of the medial condyle of the femur (arrow).

Table 9.2: Reported use of drugs for regional limb perfusion for the treatment of foals with orthopedic disease. (Perfusate volume of 10–35 mL is recommended.)

Amikacin	500 mg–1 g
Ampicillin	1–3 g
Potassium penicillin	1–2 million units
Gentamicin	200–600 mg
Timentin	1–3.1 g
Ceftazidime	1–2 g
Ceftiofur	250–500 mg
Vancomycin	150–200 mg

- Antibiotic impregnated bone cement has been shown to deliver high concentrations of antibiotics locally and constantly over a period of time. These impregnated beads can be used within joints or local soft tissue.

Prognosis

- In foals, septic arthritis typically has a favorable prognosis if the infection is identified early and the organism is sensitive to initial antibiotics.
- In cases in which the infection cannot be easily controlled or articular or subchondral bone damage is present, arthritis may develop.
- Extensive osteomyelitis can also lead to pathologic fractures due to the severe bone damage.
- Early identification and aggressive antibiotic therapy are important in the management of synovial and orthopedic infections.

Laminitis (Fig 9.108)

Laminitis is not very commonly observed in foals. It can occur due to severe inflammation of the lamina due to trauma or sepsis, or as a result of a severe decrease in blood flow to the foot. In young horses, laminitis has been observed following severe diarrhea, pneumonia, or vasculitis leading to marked limb edema. In many cases involving young horses, the degree of laminitis can be quite severe and results in marked separation of the sensitive and insensitive lamina. Sloughing of the hoof wall can occur. The diagnosis of inflammation of the foot in mild cases is often based on a thorough lameness exam.

Diagnosis

- Visual examination is diagnostic in severe cases
- Digital examination may reveal a "bounding" pulse in the digital arteries or absence of a pulse in some cases of vasculitis
- Lameness examination
- Radiology can reveal rotation of the pedal bone.

Treatment

Treatment depends on the severity of the case. Obviously in more severe cases in which hoof sloughing has occurred or there is penetration of the sole by the pedal bone, humane destruction is warranted. In milder cases the primary cause should be treated accordingly in addition to the following.

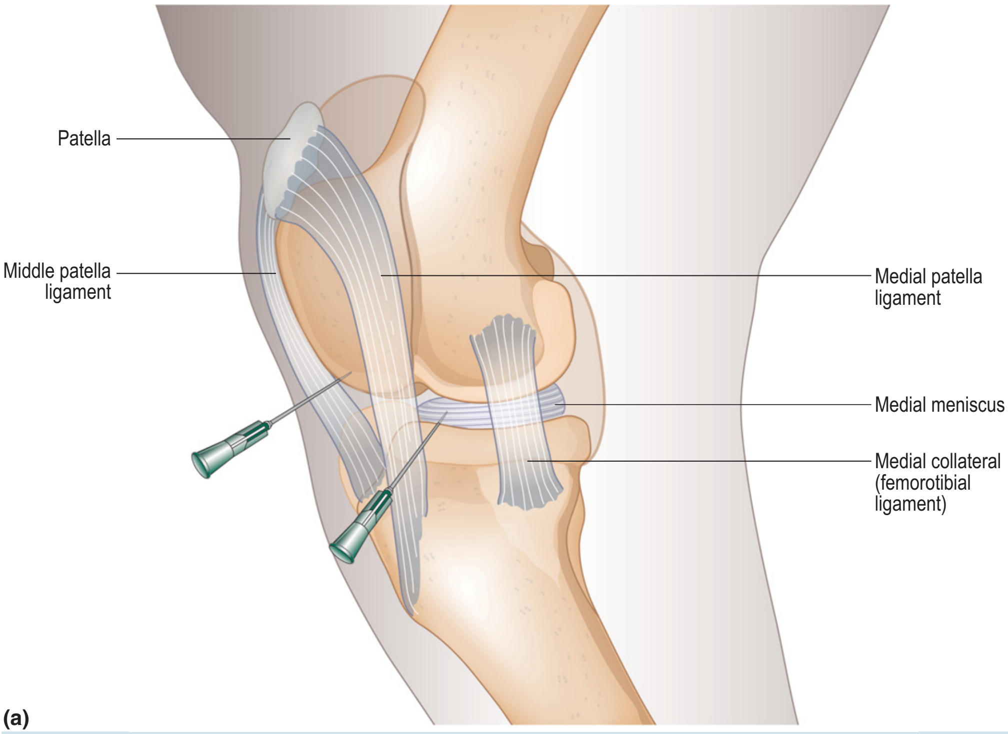
- Limit exercise:
  - stall confinement
  - ensure the stall is deeply bedded
- Medication
  - NSAIDs
  - anti-ulcer medications
- Foot care
  - foot packs to help provide cushioning
  - possible wedge shoes in the acute phase.

Fractures of the pedal bone (Figs 9.109–9.112)

Fractures involving the third phalanx are commonly observed in foals. There are eight types of fractures of the third phalanx described below.

Types of fractures

- Type I is a non-articular fracture of the palmar or plantar process of the distal phalanx
- Type II is an oblique, articular fracture of the palmar or plantar process of the distal phalanx
- Type III is a midsagittal articular fracture of the distal phalanx
- Type IV is an articular fracture involving the extensor process of the distal phalanx
- Type V is a comminuted fracture of the distal phalanx
- Type VI is a non-articular fracture involving the solar margin of the distal phalanx
- Type VII is a non-articular fracture of the palmar or plantar process of the distal phalanx, but differs from Type I in that the fracture line begins in and ends in the solar margin
- Type VIII is a non-articular fracture in the apex of P3.



(a)

(b)

Figure 9.102: (a) Technique for arthrocentesis of the stifle. The stifle joint has three pouches – the femoropatellar, and lateral and medial femorotibial. Although the medial femorotibial pouch commonly communicates with the femoropatellar pouch, for diagnostic and treatment purposes all three pouches should be evaluated separately. The femoropatellar pouch (illustrated) is entered with an 18-gauge 1.5-inch needle inserted 1 inch above the palpable tibial crest, between the middle and medial patellar ligaments. The medial femorotibial joint is entered using an 18-gauge 1.5-inch needle inserted between the medial patellar ligament and the medial femorotibial ligament. The lateral femorotibial joint is entered using an 18-gauge 1.5-inch needle directed just lateral to the lateral femoropatellar ligament just proximal to the edge of the tibia. Alternatively in many foals with septic arthritis the long digital extensor pouch, which communicates with the lateral femorotibial joint, is distended and is an easy site to enter (from Hardy J, Latimer F 2003 Orthopedic disorders in the neonatal foal. Clinical Techniques in Equine Practice 2:96–119). (b) Photographic representation of how to perform an arthrocentesis of the medial femorotibial joint.

Figure 9.103: Through and through lavage of the medial femorotibial and femoropatellar joints.

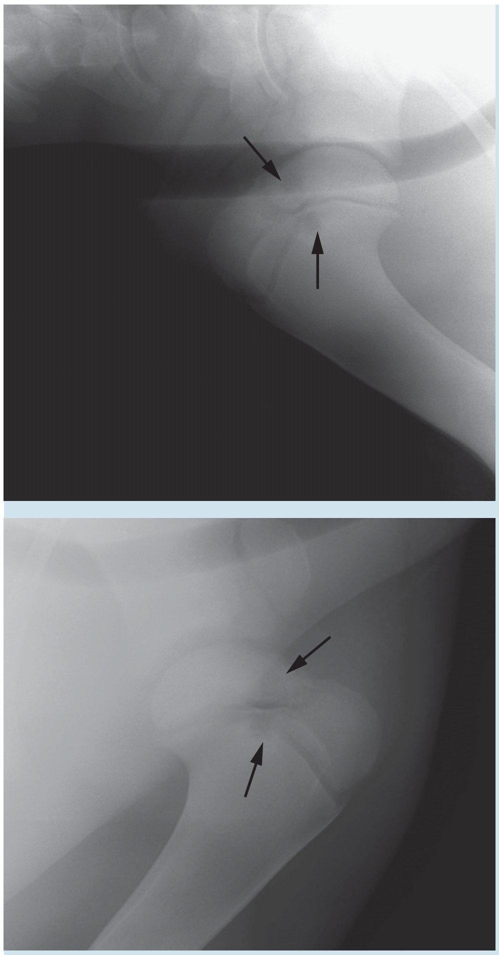


Figure 9.104: Lateral radiographs of the scapulo-humeral joint demonstrating a Type P osteoarthritis of the proximal humeral physis (arrows).

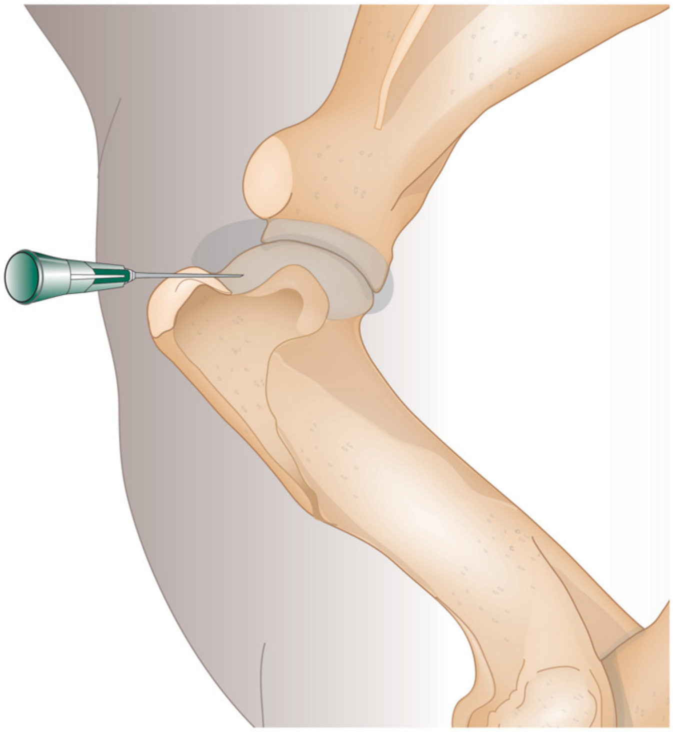


Figure 9.105: Technique for arthrocentesis of the scapulohumeral joint. An 18-gauge 3.5-inch spinal needle is used for older foals and adults. An 18-gauge 1.5-inch needle is sufficient for neonates. The needle is inserted in the palpable notch located between the cranial and caudal portions of the point of the shoulder, and directed towards the opposite elbow (from Hardy J, Latimer F 2003 Orthopedic disorders in the neonatal foal. Clinical Techniques in Equine Practice 2:96–119).

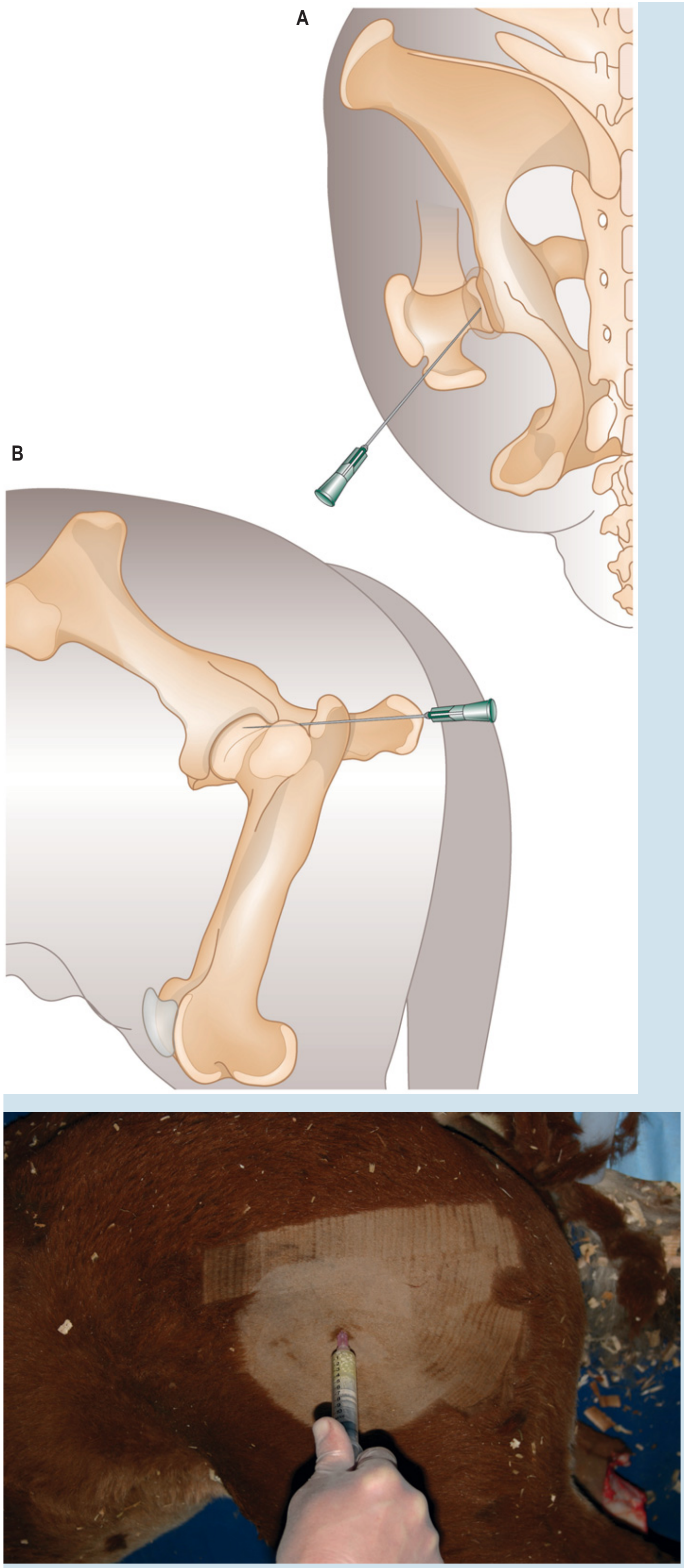


Figure 9.106: Technique for arthrocentesis of the coxofemoral joint. (a) & (b) The caudal and cranial portions of the greater trochanter are palpated, and an 18-gauge 3.5-inch spinal needle is inserted in the notch between those structures and directed approximately 25 degrees ventrally and towards the opposite stifle (from Hardy J, Latimer F 2003 Orthopedic disorders in the neonatal foal. Clinical Techniques in Equine Practice 2(1):96–119). (c) Arthrocentesis of the coxofemoral joint.

Figure 9.107 a, b & c: Radiographs from a yearling that had been treated with enrofloxacin for suspected salmonellosis as a foal. Treatment was continued after the development of joint effusion. Synovial fluid evaluations and radiographs indicated that there was no infection present. The lesions that subsequently developed (arrows) were thought ***but not proven*** to be as a result of the enrofloxacin treatment. At the time the radiographs were taken the yearling was sound but continued to have marked joint effusion.

Figure 9.108: Hoof sloughing secondary to vasculitis.

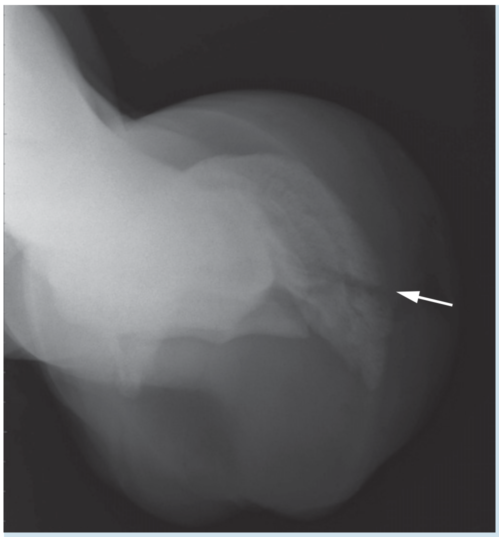


Figure 9.109: Type 2 pedal bone fracture (arrow).

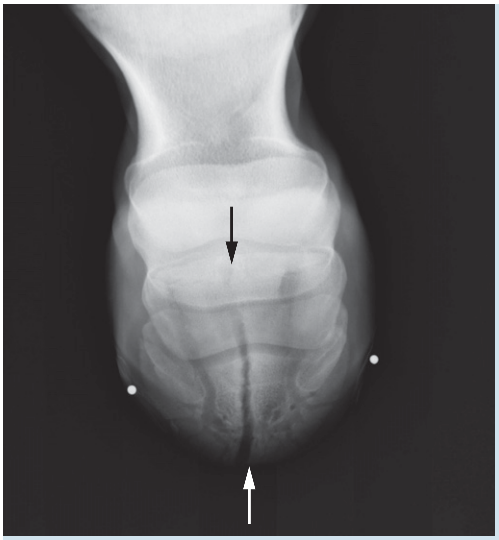


Figure 9.110: Type 3 pedal bone fracture (arrows).

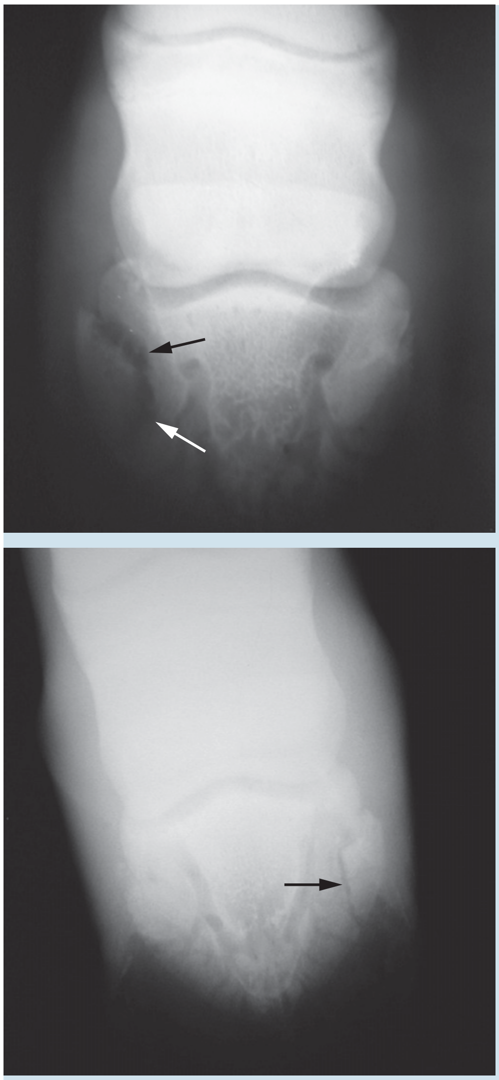


Figure 9.111: Type 6 pedal bone fracture (arrows). (Image courtesy of John Halley.)

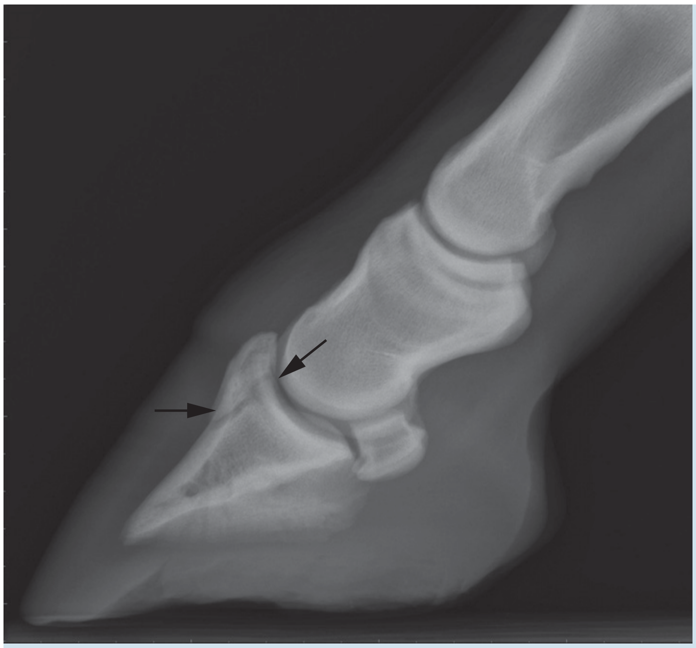


Figure 9.112: Type 4 pedal bone fracture (arrows).

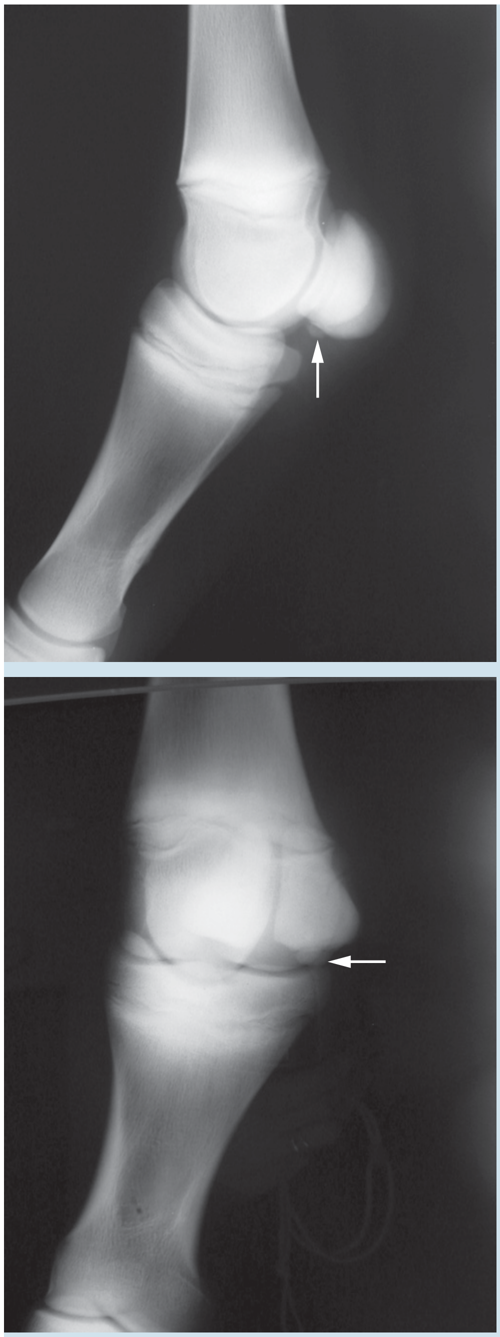


Figure 9.113: Lateral and oblique radiographs of the fetlock joint depicting basilar fractures of the sesamoid bone (arrows). (Image courtesy of John Halley.)

In foals, the most common fracture is type VII. This type of fracture generally occurs in the front limbs and usually involves the lateral wing of the third phalanx.

The fracture can involve both the lateral and medial wings of P3 and can also be bilateral in both forelimbs. Foals typically show a mild to moderate lameness in the affected limb, and will often point the affected limb.

Type VII fractures can occur following application of an acrylic extension to the foot. Though type VII is the most common fracture observed, the other types of fractures can occur infrequently.

Diagnosis

- ***Visual examination will often reveal a non-weight bearing and "pointed" limb***
- Digital examination:
  - increased heat in the foot
  - increased digital pulse
- Lameness examination:
  - able to block the lameness to the foot
- Radiology is the diagnostic tool of choice and is also useful for monitoring the healing of the fracture.

Treatment

- Limit exercise:
  - stall confinement with deep bedding for at least 2–3 weeks
  - if no longer lame, the foal may then be turned out in a small pen or paddock for a further 2–3 weeks
- Medication:
  - low dose NSAIDs
  - anti-ulcer medications
- Foot care:
  - glue on shoes can be applied
  - potentially allows the foal quicker return to a small paddock.

Fractures of the proximal sesamoids (Figs 9.113–9.115)

Fractures involving the proximal sesamoid bones can be a severe injury in foals. Fractures occur due to tension placed on the proximal sesamoid bones from the suspensory ligament and/or sesamoidean ligaments. The proximal sesamoid bones are a major component of

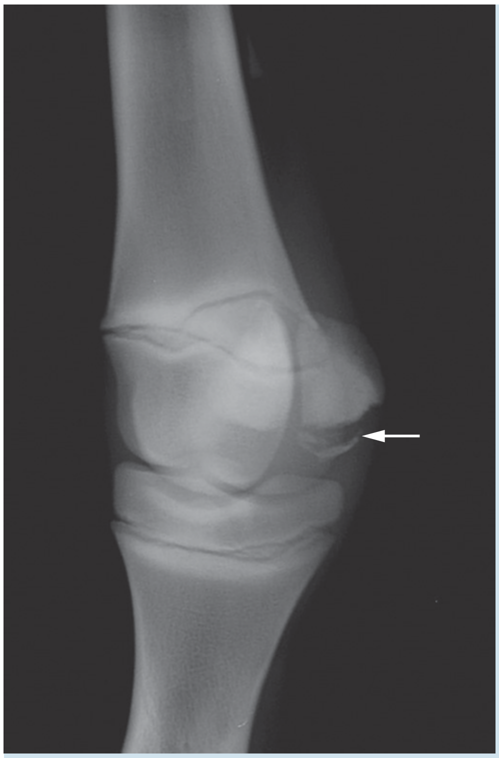


Figure 9.114: Oblique radiograph depicting a basilar fracture of the sesamoid bone (arrow)

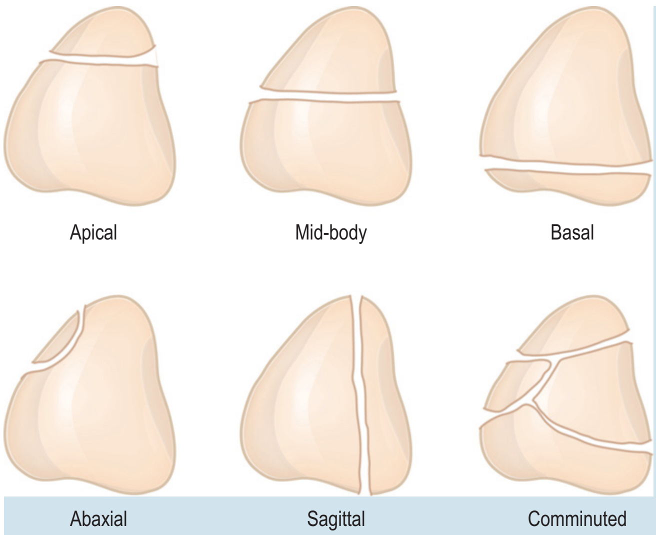


Figure 9.115: Sesamoid fracture configurations.

the equine suspensory apparatus. Disruption of both proximal sesamoid bones could lead to a disruption in the suspensory apparatus.

There are six types of proximal sesamoid bone fractures described in foals:

- apical fractures
- mid-body
- basilar
- axial
- abaxial
- comminuted.

In foals, the injury to the sesamoids is believed to occur as a result of the foal running with the mare. Excessive and/or repeated tension placed on the sesamoids results in the fracture. Fractures can also occur due to osteomyelitis of the proximal sesamoid bones.

Clinical signs

- Foals may show mild to moderate lameness initially in most cases of apical or basilar fractures
- If the fracture involves both sesamoids in one limb, the degree of lameness may be more severe
- Joint effusion of the fetlock may also be noted due to the articulation with the proximal sesamoids
- If both proximal sesamoids are affected, the fetlock may show a dropped appearance.

Diagnosis

- Visual examination:
  - joint effusion
  - swelling over the affected sesamoid
  - dropped fetlock
- Digital examination:
  - increased joint effusion
  - pain over the sesamoid
- Lameness examination
- Radiology
- Ultrasound examination.

Treatment

- Limit exercise:
  - stall confinement for 3–4 weeks followed by small pen turn-out for 3–4 weeks
- Medication:
  - Low-dose NSAIDs
  - Anti-ulcer medications
- Bandage care:
  - limb bandage for support and to help decrease swelling.

Prognosis

- Sesamoid fractures can heal with conservative management but may potentially form non-union fragments at the apex and base.
- The size of the fragments can vary. Surgical removal of the fragments can be performed as the foal matures (usually as a yearling or 2-year-old). Removal of apical fragments generally has a good prognosis for athletic soundness.
- Prognosis of apical fractures is traditionally better than with basilar fragments. Fractures of the proximal sesamoid bones in the forelimbs have traditionally been associated with a poorer prognosis than similar fractures in the hindlimbs.
- If both proximal sesamoid bones are involved and the suspensory apparatus is affected, arthritis can develop and the prognosis is poor.

Figure 9.116: OCD lesion of the sagittal ridge of the third metacarpal bone, note the flattened appearance of the saggital ridge.

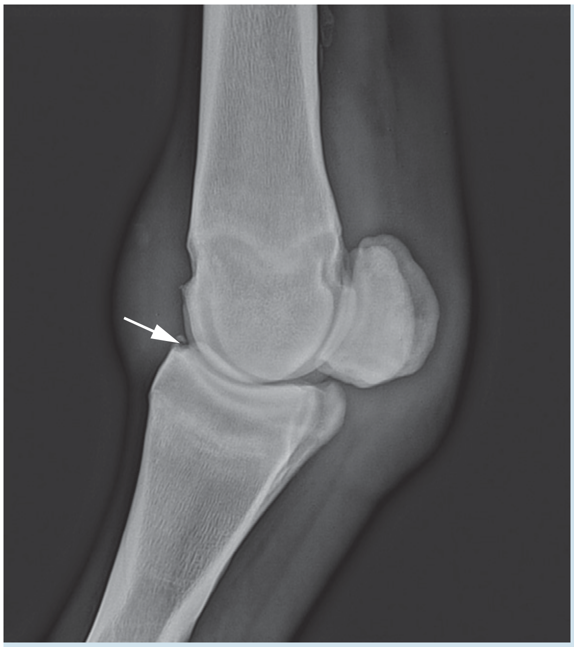


Figure 9.117: OCD lesion with chip fragment of the distal sagittal ridge of the third metacarpal bone (arrow).

Figure 9.118: OCD lesion with chip fragment of the plantar process of P1 (arrow).

Figure 9.119: Tarsal (hock) effusion in a weanling with OCD.

- In cases of severe disruption of the suspensory apparatus, surgical arthrodesis of the fetlock is potentially required.

Osteochondrosis (Figs 9.116–9.130)

Osteochondrosis has often been defined as failure of endochondral ossification resulting in either osteochondritis dissecans (OCD) or subchondral bone cysts. Endochondral ossification occurs within the epiphysis, physis and cuboidal bones of the carpus and tarsus. Endochondral ossification involves growing cartilage which is systematically replaced by bone. The cartilage in the physis is composed of numerous zones or layers. The zones at the physis include: resting, proliferative, hypertrophic, and calcified. At the articular surface, the zones are similar but the articular surface is called the superficial zone of cartilage.

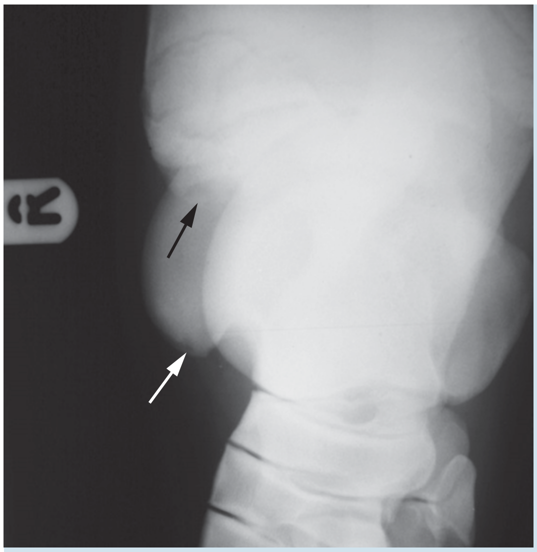


Figure 9.120: Dorsomedial-plantarolateral radiographic view of the hock demonstrating an OCD lesion of the distal lateral trochlear ridge of the talus (white arrow) and the cranial intermediate ridge of the distal tibia (black arrow).

Figure 9.122: Dorsomedial-plantarolateral radiograph of the hock demonstrating an OCD lesion of the cranial intermediate ridge of the distal tibia (arrow).

Figure 9.121: Dorsomedial-plantarolateral radiographic views of the hock demonstrating OCD lesions of the distal lateral trochlear ridge of the talus (arrow). OCD fragments at this site can be large but the prognosis is good following surgical removal. Smaller fragments that do not result in joint effusion or lameness do not always require removal.

Figure 9.123: Effusion of the femoro-tibial joint in a foal with OCD.

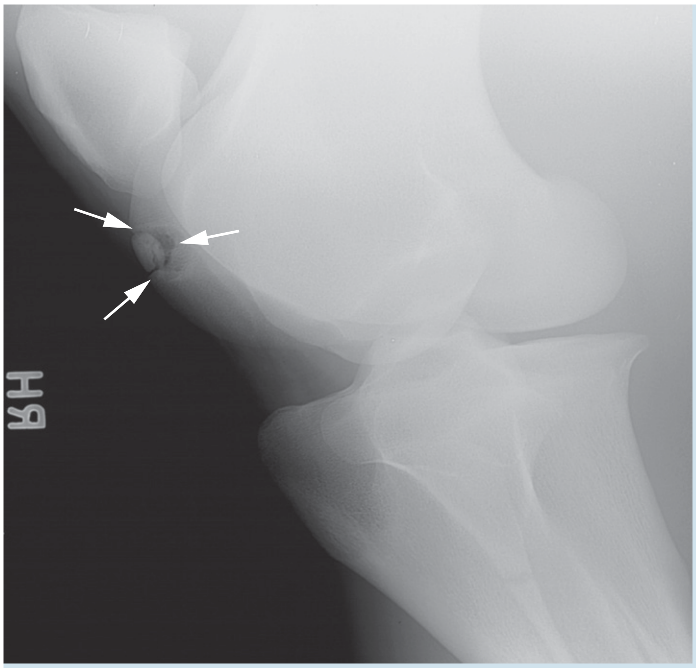


Figure 9.124: Radiograph of the stifle demonstrating an OCD lesion of the lateral trochlear ridge of the femur (arrows).

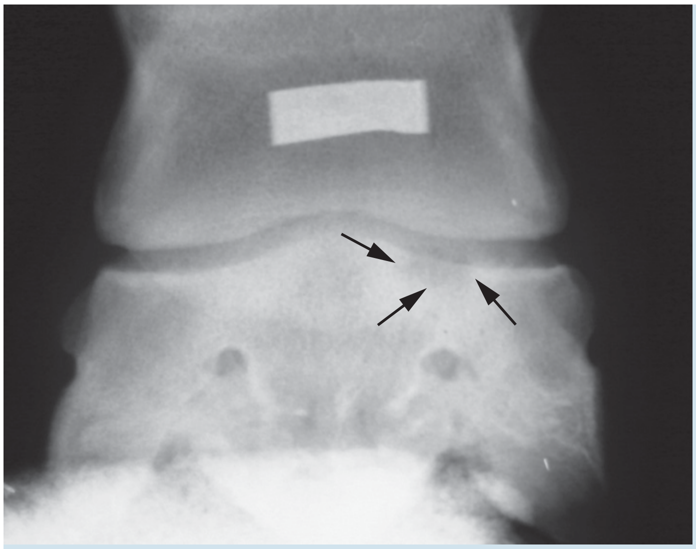


Figure 9.125: Subchondral bone cyst of P3 (arrows).

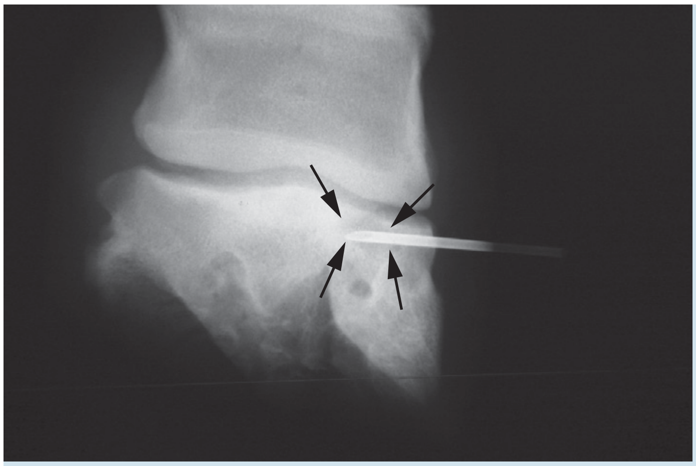


Figure 9.126: Intraoperative radiograph of needle placement in the cyst (arrows).

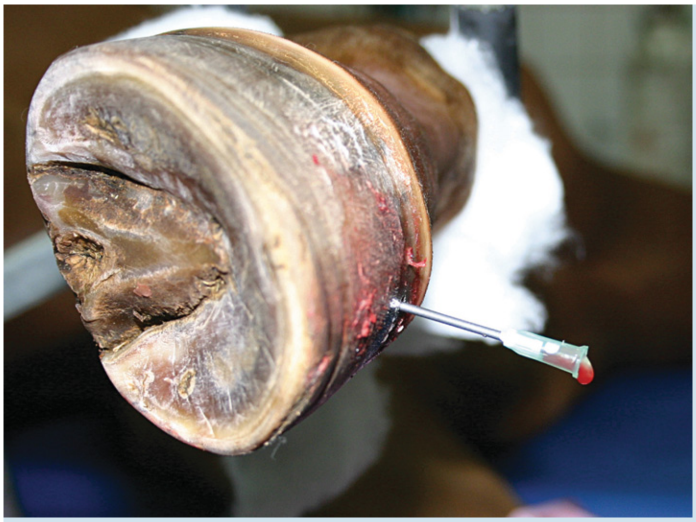


Figure 9.127: Synovial fluid was obtained from the site indicating a communication with the distal interphalangeal joint.

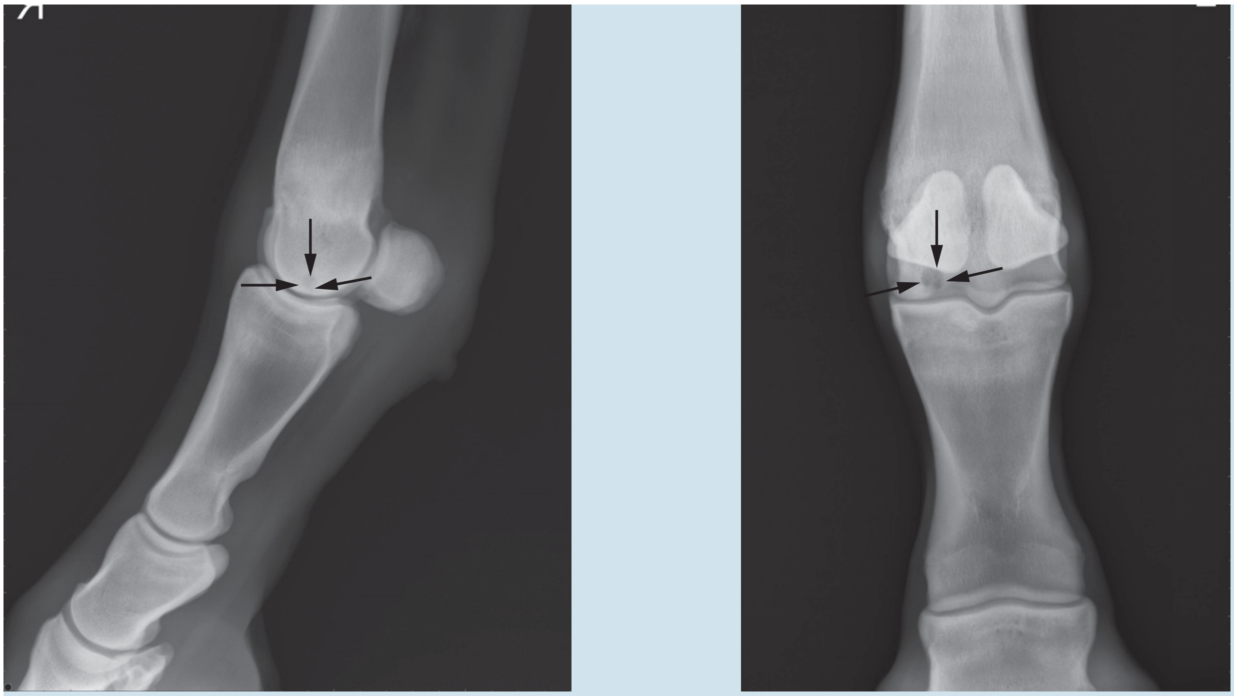


Figure 9.128: Lateral and dorsopalmar radiographs of a fetlock joint demonstrating a subchondral bone cyst (arrows).

Figure 9.129: Cyst lesion of the distal metaphysis of the radius (arrows).

Besides defects in the process of endochondral ossification, other causes have been suggested including loss of blood supply, genetic, gender specific, dietary, endocrine, biomechanical, traumatic, and toxic. Body size and growth rate have also been proposed as being a potential cause of the development of osteochondrosis. A study evaluating Standardbred foals found that a positive relationship existed between the body weights of foals at birth, body weight during the growth period, average daily weight gain, skeletal frame size and development of osteochondrosis lesions.

Nutritional factors have also been suspected of causing osteochondrosis. High energy diets have long been thought to be associated with causing defects in the development of articular and physeal cartilage. Along with high-energy diets, hyperinsulinemia has been associated to the development of osteochondrosis. Studies evaluating diets high in phosphorous, calcium and excessive energy found an association of cartilage dysplasia with high phosphorous and high energy. High calcium did not cause cartilage dysplasia or prevent the occurrence of cartilage dysplasia. Low copper diets have been linked to the occurrence of osteochondrosis. Copper is important in the collagen network of cartilage and bone. Copper antagonists such as zinc and molybdenum can also lead to cartilage dysplasia.

Involvement of the small blood vessels passing through cartilage canals in the epiphysis has been proposed as a possible link to osteochondrosis. Ischemic necrosis of the articular cartilage due to potential damage of these vessels has been proposed.

The level of exercise young horses receive has also been implicated in the development of osteochondrosis. Excessive exercise in young foals has been speculated as a potential cause. However a decrease in the level of exercise has also been found to potentially increase the incidence of osteochondritis. One study found no correlation between exercise and osteochondrosis, but did propose that different levels of exercise could change the presentation and degree of osteochondrosis. One study found that low level exercise caused potential lesions on the medial femoral condyle, and that foals exercised developed lesions on the femoral trochlear ridge. This study also showed a tendency for the lesions to be more severe in foals with limited exercise.

Trauma and biomechanical stresses have been proposed as causing osteochondrosis. Biomechanical forces can potentially cause an osteochondritis lesion to become an osteochondritis dissecans lesion.

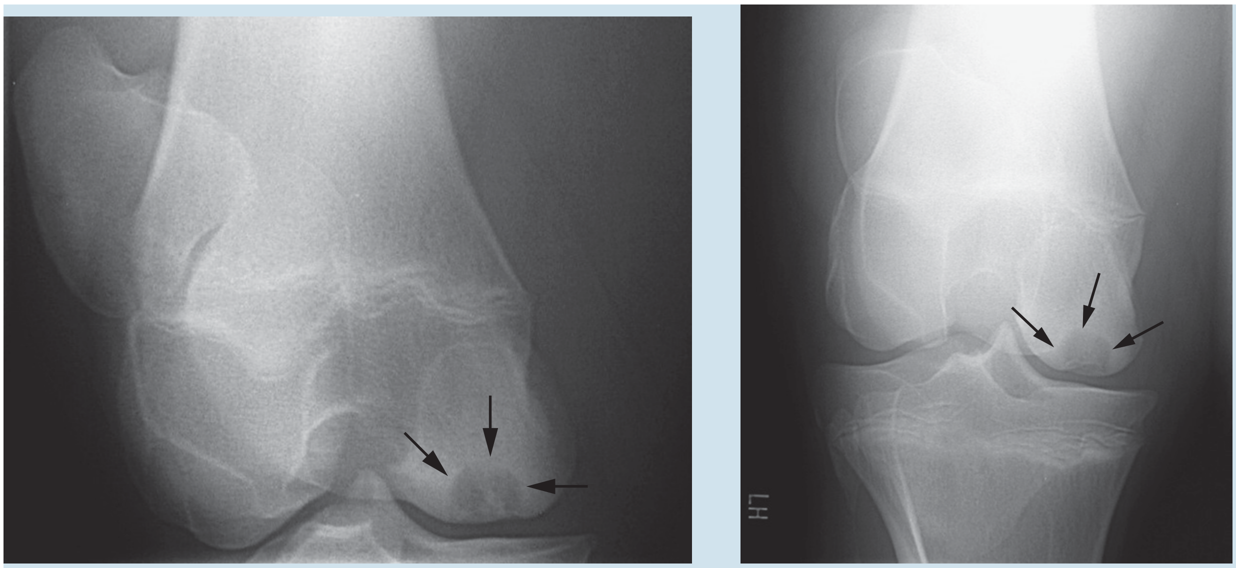


Figure 9.130: Subchondral bone cysts of the femorotibial (arrows).

Table 9.3: Osteochondritis dissecans (OCD)

JOINT	LOCATION
Fetlock	Mid-Sagittal Ridge
	Dorsal Proximal Medial/Lateral P1
	Palmar/Plantar P1
Tarsus	Distal Intermediate Ridge of Tibia
	Medial Malleolus
	Lateral Trochlear Ridge
	Medial Trochlear Ridge
Stifle	Lateral Trochlear Ridge
	Medial Trochlear Ridge
	Distal Aspect of Patella
Shoulder	Glenoid Cavity
	Humeral Head
Elbow	Lateral/Medial Humeral Condyle (Uncommon)

Table 9.4: Subchondral bone cysts

JOINT	LOCATION
Stifle	Medial Femoral Condyle
	Proximal Tibia
Fetlock	Distal Condyle of Metacarpus/Metatarsus
Pastern	Distal P1
Shoulder	Glenoid
	Humeral Head
Elbow	Proximal Radius
Coffin	Third Phalanx
Carpus	Ulna, Radial Carpal Bones (most commonly)

Subchondral bone cysts are now speculated to originate from possible mechanical insults when compared to osteochondritis.

Osteochondrosis commonly occurs in the tarsus, stifle, and fetlock joints. Though uncommon, osteochondrosis can occur in the scapulohumeral, elbow, and hip joints. Tables 9.3 and 9.4 list the most common locations of lesions within each joint.

Lesions can potentially regress. In the hock, 5 months has been used as a possible cut-off for lesions regressing. After 5 months, lesions will typically not regress. In the stifle, 8 months is the cut-off for a lesion regression involving the lateral trochlear ridge.

Clinical signs

- Clinical signs can include joint effusion and lameness. However, in certain situations lameness and joint effusion may not be present.
- Joint effusion is more common with a dissecting lesion.
- Lameness can be present with both dissecting lesions and cysts, but cysts can often be asymptomatic until horses begin training.
- ***It is very important to examine the contralateral joint for the presence of an osteochondral lesion as lesions are frequently bilateral.***
- Radiographic examination of the joint(s) in questions will often provide a definitive diagnosis. In some cases, joint effusion is present but radiographs show no significant findings. In these cases, serial radiographs over 4–6-week intervals may be required to confirm the absence or presence of an osteochondritis lesion.
- Diagnosis of osteochondritis is often easily made based on signalment, history, clinical signs and radiographic findings.
- Ultrasound examination of affected joints can also help to evaluate and confirm the presence of a cartilaginous flap.

Treatment

Treatment of osteochondral lesions involves conservative or surgical management.

- Conservative management involves limiting exercise and chondroprotective therapy and is indicated in young foals and those that do not have any effusion or lameness associated with their OCD lesion(s).
- Surgical management of osteochondritis dissecans typically involves surgical debridement of the fragment and the fragment bed. Surgical

management of subchondral bone cysts has included debridement, cartilage replacement, packing the cyst with bone marrow, or most commonly injection of corticosteroids.

- Surgical treatment should be considered for foals with joint effusion and lameness. Prophylactic removal of OCD fragments may also be considered in young horses intended for sale.

Prognosis

Prognosis for osteochondritis dissecans and subchondral bone cysts vary based on location, number of joints involved, size of the lesion, presence of osteoarthritis, and the degree of lameness.

Fetlock

Many fetlock lesions can improve radiographically over time with conservative management. Surgical treatment should be considered in foals or young horses less than 18 months of age that demonstrate joint effusion and lameness. Lesions of the palmar/plantar aspect of P1 do not often require surgery.

Hock (tarsus)

The distal intermediate ridge of the tibia is the most common site of lesions and these are rarely associated with effusion and lameness.

Lesions of the medial malleolus are more likely to result in joint effusion and lameness requiring surgical treatment.

Lesions of the lateral trochlear ridge can be quite variable in size but if large may result in effusion and lameness.

Most medial trochlear ridge lesions are incidental findings and do not require treatment.

Stifle (femoropatellar joint)

The lateral trochlear ridge is the most common site of lesions in the femoropatellar joint. A number of femoropatellar OCD lesions can heal with conservative therapy. One recommendation is that all lesions greater than 2 cm in length and 5 mm in depth, or any lesion that contains osseus densities in the presence of synovial effusion should be treated surgically. In most cases surgery is not recommended prior to 8 months of age. When the changes are regarded as severe, with a poor prognosis for athletic function, surgery is not recommended.

Shoulder and elbow

OCD lesions of the shoulder and elbow are normally only recognized when associated with lameness. The lameness in these cases may develop insidiously or acutely and identifying the site of the lesion is often difficult. Surgery should only be considered in foals less than 8–10 months of age if they are intractably lame, as some cases may improve with rest. Radiography may also underestimate the size of the lesions making pre-surgical assessments of future athletic function difficult.

Epiphysitis and physitis (Figs 9.131 & 9.133)

Physitis is a common problem affecting foals. Most commonly the disease affects horses 3–12 months of age, but horses up to 2 years of age can be affected. Typically it involves fine bone breeds on a high

Figure 9.131: Epiphysitis of the distal third metacarpal bone.

Figure 9.132: Radiograph of the fetlock (metacarpaphalangeal) joint from the foal in Fig 9.131 demonstrating widening and sclerosis of the physis.

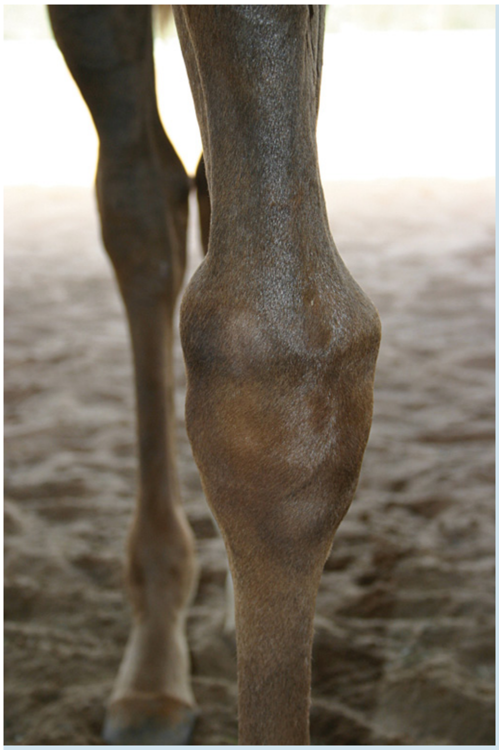


Figure 9.133: Epiphysitis of the distal radius.

plane of nutrition. The most common sites of physitis include the distal radius, distal metacarpus/metatarsus, and distal tibia.

Clinical signs and diagnosis

- Visual examination reveals swelling involving the distal radial physis, distal physis of the metacarpus/metatarsus, and distal physis of the tibia.
- Digital palpation reveals heat and pain over the affected physis.
- Diagnosis can be made on clinical signs, but radiographic examination can also be performed. Radiographic examination can reveal sclerosis of the physis and possible periosteal callus formation.

Treatment

- Treatment of physitis involves lowering the plane of nutrition. This may involve preventing access to the mares feed, muzzling the foal if the mare is a heavy milk producer (e.g some nurse mares) or the foal could be weaned.
- Anti-inflammatories and anti-ulcer medication should be considered in all cases.
- The use of topically-applied poultices has anecdotal reports of benefit.
- In cases involving an angular limb deformity, the affected limb should be trimmed correctly. In severe cases, transphyseal bridging procedures may be required.

Prognosis

Prognosis is good if the degree of physitis is mild and there is no evidence of an angular limb deformity.

Figure 9.134: Splint bone fracture (arrow) with associated large callus..

Splint bone fracture (Figs 9.134 & 9.135)

Fractures of the splint bones generally occur due to a traumatic insult. In foals, fractures involving the splints are not very common. The lateral splint is more often involved.

Clinical signs and diagnosis

- Visual examination and digital palpation are important in the diagnosis
- A firm painful swelling is often observed with a fractured splint bone
- Radiographs are important in diagnosis and in determining the best method of treatment; it is also important to rule out focal exostosis from a fracture
- A periosteal reaction over the splint bone due to an injury causing inflammation of the interosseous ligament can also cause a firm soft tissue swelling

Treatment

If the splint is fractured, medical or surgical management can be instituted. Treatment options vary based on the splint involved, location on the splint bone, open or closed fracture, and degree of displacement. Surgical intervention is used in cases with marked displacement and contaminated fractures.

- Medical management:
  - half-limb bandages
  - limited turn-out
  - repeat radiographic examination
- Surgical management:
  - remove distal fragment

A

B

Figure 9.135: (a) Splint bone fracture (arrow). (b) Same foal 3 weeks later, showing a large callus at the fracture site.

- surgical stabilization of the proximal and distal fragments
- surgical debridement of the fracture but the distal fragment is not removed.

Prognosis

- Prognosis is generally good with both medically and surgically managed cases.
- If the splint bone is the second metacarpal bone (medial splint bone) and the fracture is proximal, surgical removal of the distal fragment could lead to instability of the proximal fragment. This is due to the articulation of the proximal aspect of the splint with the second and third carpal bones. The entire fourth metacarpal bone can be removed in select cases.
- If the fracture is open and is located close to either the carpus or tarsus articulation, the prognosis may be poor due to the possibility of joint sepsis.

Salter–Harris fractures / injuries (Fig 9.136)

Salter–Harris fractures are fractures that involve the physis. The six types of fractures and their respective treatments are listed below.

Type I. The fracture is through the physis with no involvement of the epiphysis or metaphysis. The germinal cells remain on the epiphyseal side. Closed reduction using external coaptation may be used; however, if there is significant displacement, surgical internal fixation is also required.

Type II. The fracture is through one side of the physis and also breaks through the metaphysis. This is a common fracture observed in foals. This fracture is seen in the proximal tibia, distal femur and distal metacarpus/metatarsus. Closed reduction using external coaptation may be used; however, if there is significant displacement, surgical internal fixation is also required.

Type III. The fracture courses through the one side of the physis and then breaks through the epiphysis into the joint. This fracture is articular, and is not very common in foals. Surgical reduction and internal fixation is required to provide accurate alignment of the articular fracture. External coaptation is used to provide further stability.

Type IV. The fracture extends from the joint surface through the epiphysis and physis and then breaks through the same side in the metaphysis. The fracture is articular, and is also rare in the foal. Surgical reduction and internal fixation is required to provide accurate alignment of the articular fracture. External coaptation is used to provide further stability.

Type V. This injury is due to a crushing or collapsing of the growth plate involving either the medial or lateral side of the growth plate. This is possibly observed in foals involving the medial aspect of the distal metacarpus. Collapse of the medial side of the distal metacarpus growth plate is possibly the reason for the fetlock varus deformities noted in foals at 3–4 months of age.

Type VI. This type of injury is a result of the development of a periosteal bridge between the metaphysis and epiphysis. This results in growth retardation on the side with the periosteal bridge. This type of injury has been noted following removal of transphyseal bridge implants such as staples and screw and wires. Placement of a transphyseal bridge on the opposite side can help correct growth discrepancies associated with this type of injury. Surgical removal of the periosteal bridge could also be performed, but there is the potential for the bridge to reform.

Prognosis for Salter–Harris fractures depends on the type of fracture and articular involvement.

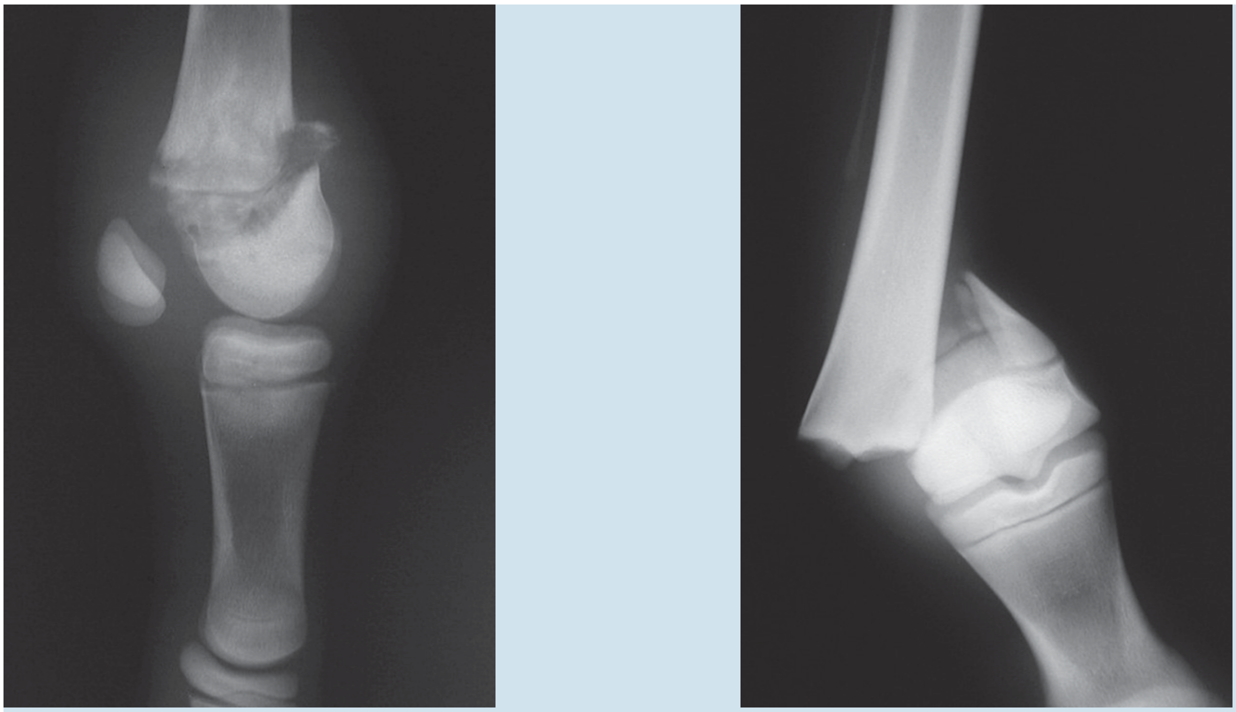


Figure 9.136: (a) Salter–Harris fracture Type I in a foal with septic osteomyelitis. (b) Salter–Harris fracture Type II.

Ischemic necrosis of the medial femoral condyle (Figs 9.137 & 9.138)

This condition is an infrequently seen complication of septicemia. It is thought to result from an ischemic process but the exact etiology is not understood. Affected foals present with moderate to severe lameness and marked effusion of both femorotibial and femoropatellar joints, between 3–4 weeks of age. Radiography reveals thin fragments displaced from the medial condyle into the femorotibial pouch with minimal subchondral bone changes. Analysis of the synovial fluid generally fails to indicate osteoarthritis. Any foal with a history of septicemia that presents with lameness and effusion of the stifle should have plantar–dorsal radiographs taken in addition to lateral views. Postmortem examination of these foals reveals a large area of exposed subchondral bone with separation of osteochondral fragments over 60–70% of the medial condyle. Prognosis for these cases is grave due to the extent and severity of the lesions.

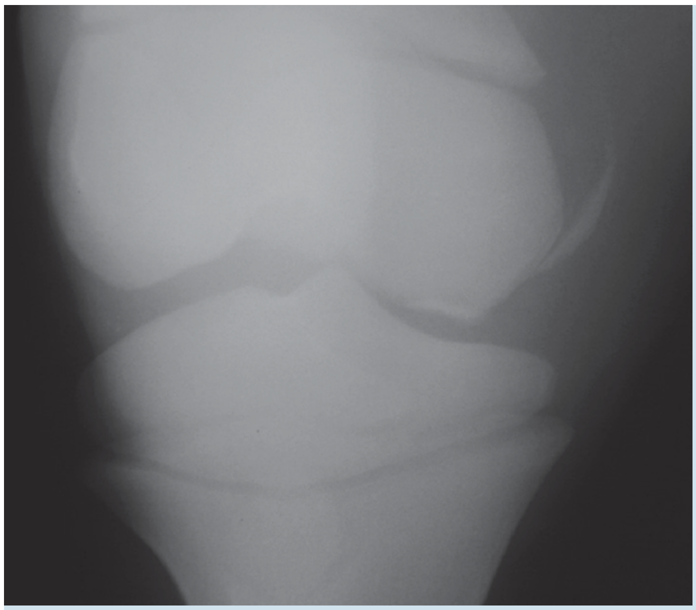


Figure 9.137: Caudo-cranial radiograph from a foal with ischemic necrosis of the medial femoral condyle. Note the thin fragment of bone that has become separated from the medial condyle.

A

B

Figure 9.138: Same foal as Fig 9.137 at postmortem. Note the separation of the cartilage from a large area of the femoral condyle. In (b) note the thin fragment of bone attached to the cartilage that had been imaged radiographically.

Recommended reading

Auer JA 1999 Angular limb deformities. In: Auer JA, Stick JA (eds) Equine surgery. WB Saunders, Philadelphia, p 736–752

Hance SR, Schneider RK, Embertson RM et al 1993 Lesions of the caudal aspect of the femoral condyles in foals: 20 cases (1980–1990). Journal of the American Veterinary Medical Association 202(4):637–646

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Nixon AJ 1999 The phalanges and metacarpometa-tarsophalangeal joint. In: Auer JA, Stick JA (eds) Equine surgery. WB Saunders, Philadelphia, p 792–809

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CHAPTER 10

The liver, peritoneum and spleen

Thomas J. Divers DVM, DACVIM, DACVECC

Assessment of liver function and diagnosis of liver disorders is commonly based on liver-specific laboratory tests and information gained through biopsies in addition to history, clinical signs and complete blood counts.

Liver-specific laboratory tests

Sorbitol dehydrogenase (SDH) is widely used to evaluate acute liver disease in the horse. It has a very short half-life with values returning to baseline within 3–5 days after a transient liver insult. It is a hepatocellular enzyme. Analysis should be done within several hours of collection; if transit time is prolonged then storage of the serum in a refrigerator or freezer would be warranted.

γ-Glutamyltransferase (GGT) is primarily associated with the microsomal membranes in the biliary epithelium. The half life of GGT is 3 days and is considered stable in serum for 2 days at room temperature. GGT is usually elevated with diseases associated with the biliary tract. Foals will normally have mild elevations of GGT for the first 3–4 weeks of life.

Bilirubin

Bilirubin levels are not a sensitive indicator of liver disease in the foal. Total bilirubin concentration in the serum is a combination of unconjugated (indirect) and conjugated (direct) bilirubin. One must therefore determine the concentrations of unconjugated and conjugated bilirubin when evaluating liver disease.

- Unconjugated bilirubin levels can be elevated with anorexia or hemolysis. Neonates normally have more unconjugated bilirubin levels than adults. This is most likely caused by the turnover of fetal hemoglobin to adult hemoglobin and the deficiency of liver binding and conjugating enzymes compared to the adult.
- Conjugated bilirubin elevations are more indicative of hepatic disease (hepatocellular or cholestasis). Conjugated bilirubin is water soluble and can be detected in the urine when blood concentrations are sufficiently increased to surpass the renal threshold.

Bile acids

- The liver normally removes 90% of the bile acids from the enterohepatic circulation. Elevations in bile acids would indicate liver failure (normal 0–30 μmol/L). A variety of diseases can cause elevated serum bile acids which include:
  - Portosystemic shunt
  - Failure of the liver to remove the bile acids
  - Failure of the hepatocytes to conjugate the bile acids for excretion
  - Failure of excretion with secondary regurgitation of the bile acids into the blood.

Blood ammonia

- The liver is responsible for removing ammonia from circulation and converting it to urea for renal excretion.
- Increases in ammonia levels or decreased blood urea nitrogen (BUN) concentrations would strongly indicate liver dysfunction
  - Gastrointestinal disease with ammonia-generating bacteria could cause excessive ammonia production. This could result in higher than normal blood ammonia levels.
- Samples are collected in an EDTA tube and must be cooled immediately on ice. Refrigerated whole blood is stable for up to 6 hours without significant increase in ammonia concentrations.
- Normal range for blood ammonia is 0–63 mg/dl, however normal values vary between labs. It is best to call the lab that will be running the test for their normal range. Most labs will also request an age matched control.

Liver biopsy

A liver biopsy can yield important diagnostic, prognostic and therapeutic information. The biopsy is performed on the right side between the 12th and 15th intercostal spaces. An ultrasound should be used to visually assess the depth and location of the liver. The area is then clipped, aseptically prepared and injected with 1 cc of 2% lidocaine. A stab incision using a No. 15 blade is made before inserting a Tru-Cut 14 gauge 16 cm biopsy instrument that is directed cranially/ventrally into the liver.

- Specimens are directly placed in formalin for histopathologic evaluation.
- Specimens should also be collected into a sterile vial for cultures.

Complications associated with a liver biopsy may include:

- Hemorrhage. Although frequently cited in literature this is a rare occurrence in practice. Clotting profile may be performed prior to the biopsy to assess the risk of hemorrhage
- Colonic puncture with peritonitis
- Pneumothorax
- Systemic dissemination of the infectious hepatitis.

Figure 10.1: Comatose, 5-week-old TB foal with Tyzzer's disease.

Figure 10.2: Bilirubinuria in a foal with Tyzzer's disease.

Hepatic disorders

Liver disease in foals is uncommon; however *increases* ***in liver enzymes and bilirubin concentration can frequently be seen in septicemic neonates.***

- Differentials for liver disease in neonatal foals include:
  - Tyzzer's disease
  - equine herpes virus 1 infection
  - hepatic damage secondary to neonatal isoerythrolysis
  - biliary obstruction secondary to duodenal ulceration
  - umbilical vein infections
  - portal vein thrombosis
  - toxic hepatopathy.
- Older foals (6–12 weeks) may show neurological signs as a result of:
  - portosystemic shunt
  - congenital deficiency of ammonia metabolism (Morgan's)
  - hepatic lipidosis.

Tyzzer's disease (Figs 10.1–10.3)

History

Tyzzer's disease is a sporadic cause of bacteremia and liver disease with failure in 6–42-day-old foals. Although not considered to be contagious, it may be endemic on some farms, possibly associated with heavier environmental contamination. The causative agent is *Clostridium piliforme*, a spore-forming, soil and manure borne, Gram-negative obligate intracellular bacterium. Outbreaks of this disease have been noted in rabbits and rodents.

Clinical signs

- The onset of clinical signs is usually peracute and typically fatal in 2–48 hours
- Signs include depression, tachypnea, tachycardia, fever, icterus, injected mucous membranes, diarrhea, weakness, and bilirubinuria
- Many cases are found recumbent, blind and comatose

Differential diagnosis

- The signs associated with sepsis (depression, fever, tachycardia, tachypnea, etc.) could be seen in similar age foals due to:
  - acute pneumonia (*R. equi*) (in foals >3 weeks)
  - acute peritonitis from a ruptured abscess (*R. equi*, *Streptococcus equi*) or ruptured bowel (gastric ulcer)
  - acute salmonellosis.

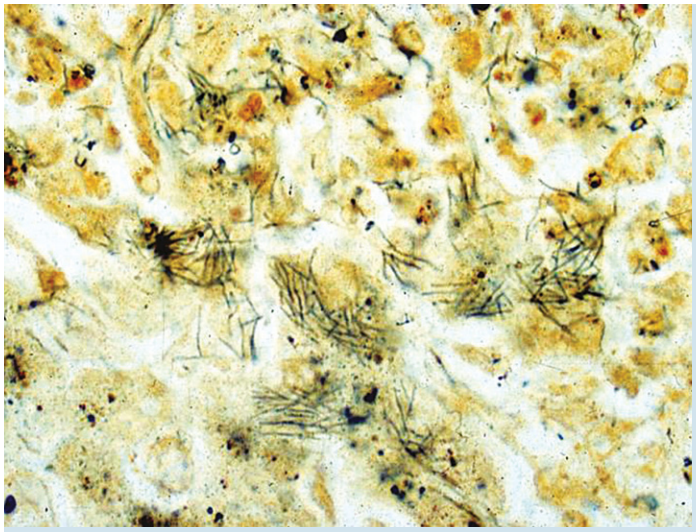


Figure 10.3: Silver stain of a section of liver from foal with Tyzzer's disease. *Clostridium piliforme* organisms are seen as large rods.

- The prominent neurological signs, e.g. coma and blindness, could be found with:
  - bacterial or viral meningitis
  - *Streptococcus equi* abscess in the brain.
- Other causes of hepatic failure in this age group are; herpes virus 1 hepatitis (possible but rare in this age group), portosystemic shunts and idiopathic causes of liver failure.

Diagnosis

- A tentative diagnosis is based upon age of the foal, clinical signs and laboratory findings which include severe metabolic acidosis, hypoglycemia, and increases in both direct and indirect bilirubin, hepatic enzymes, and bile acids in the serum.
- Frequently, there is a degenerative left shift on the leukogram and a thrombocytopenia.
- Liver biopsy is rarely performed because the clinical signs and laboratory findings are so characteristic of Tyzzer's disease that a biopsy is not required for a tentative diagnosis and, in fact, due to the frequent occurrence of thrombocytopenia, a biopsy may be contraindicated.
- Gross pathologic findings include hepatomegaly with multifocal areas of light discoloration.
- The diagnosis is usually confirmed histologically on autopsy. *Clostridium piliforme* is poorly visualized with routine staining methods and detection is greatly facilitated by the use of silver stains such as Warthin-Starry stain.
- PCR on a liver sample has recently been used to diagnose Tyzzer's disease in a 16-day-old Paint Horse filly at the University of California Davis Veterinary School.

Treatment

- There has been one confirmed case of Tyzzer's disease that survived in addition to several suspected cases.
- The treatment would focus on treatment of the septic shock. Crystalloids with dextrose and plasma should be given intravenously, followed by vasopressor therapy if needed to improve blood pressure and urine production.
- The severe hepatic necrosis results in impaired metabolism of nutrients and vitamins, highlighting the importance of parenteral nutrition.
- Flunixin meglumine should be given to decrease fever and to inhibit inflammatory prostanoids.
- If the metabolic acidosis does not improve and remains <7.25 pH following fluid therapy, sodium bicarbonate should be given.
- If the foal is demonstrating obvious signs of hepatoencephalopathy, mannitol can be administered IV in addition to lactulose (per rectum or orally).
- Antibiotics (penicillin and aminoglycosides) or tetracycline should be given intravenously.

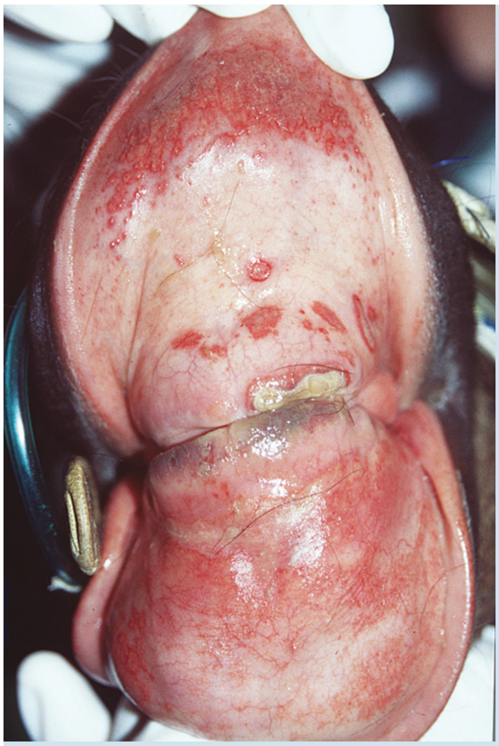


Figure 10.4: Thoroughbred colt with EHV-1 infection/vasculitis. Note the petechial and ecchymotic hemorrhages.

Equine herpes virus 1 hepatitis (Figs 10.4–10.10) (see Chapter 1 p. 11; Chapter 5 p. 147)

History and clinical signs

Mares that become infected with EHV-1 late in gestation may deliver a live foal that is jaundiced and weak at birth or the foal may appear normal at birth but develop icterus, vasculitis, respiratory distress, and, rarely, diarrhea within the first 5 days of life.

Differential diagnosis

Icterus is a common finding in any septic foal, so any and all causes of septicemia, especially those with evidence of pulmonary dysfunction,

Figure 10.5: Same colt as in Fig 10.4 at necropsy demonstrating vasculitis in several organs.

Figure 10.8: Liver from a foal that died of EHV-1 showing focal areas of hepatic necrosis.

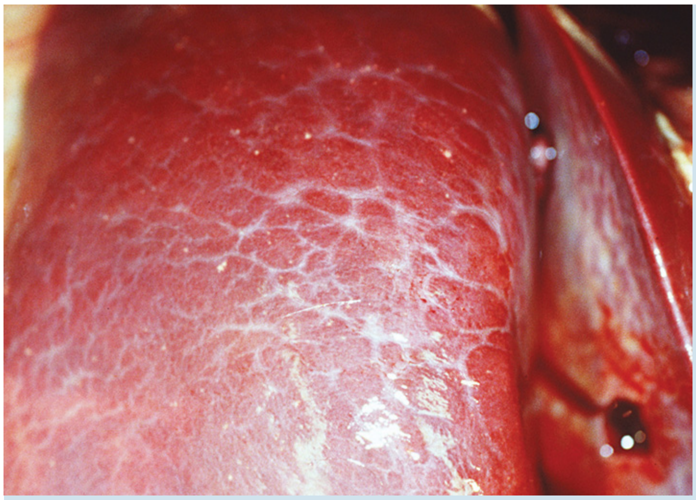
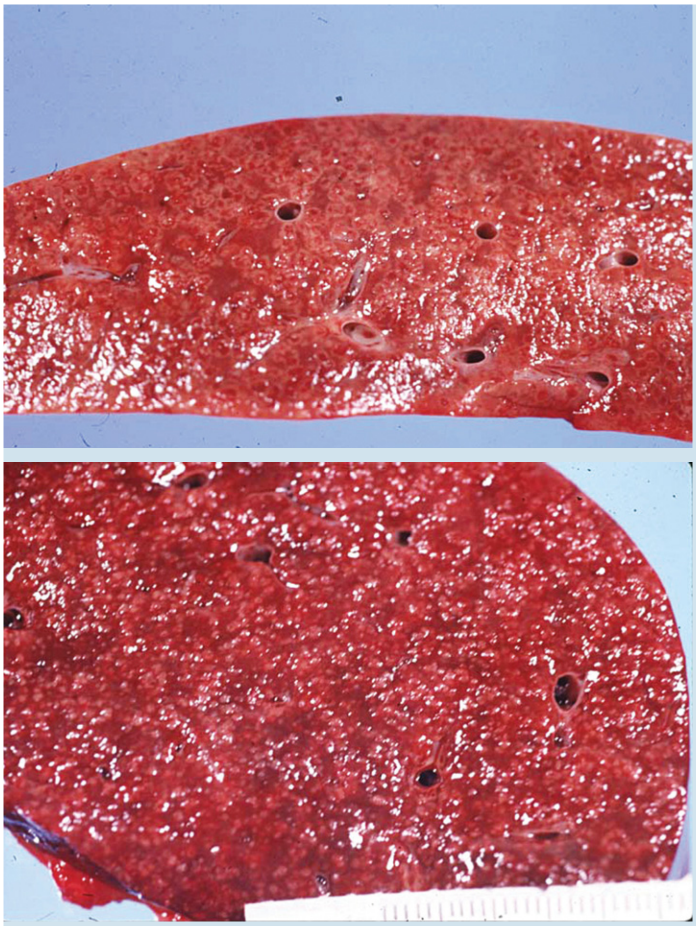


Figure 10.6: Necropsy of a foal with EHV-1 infection. Note marked pneumonia with nodular lesions on the surface of the lung.



Figures 10.9 & 10.10: Livers from 3- and 5-day-old foals that died from *Actinobacillus* and *Streptococcus* bacteremia. Appearance of liver and age of foal can be easily mistaken for EHV-1 infection.

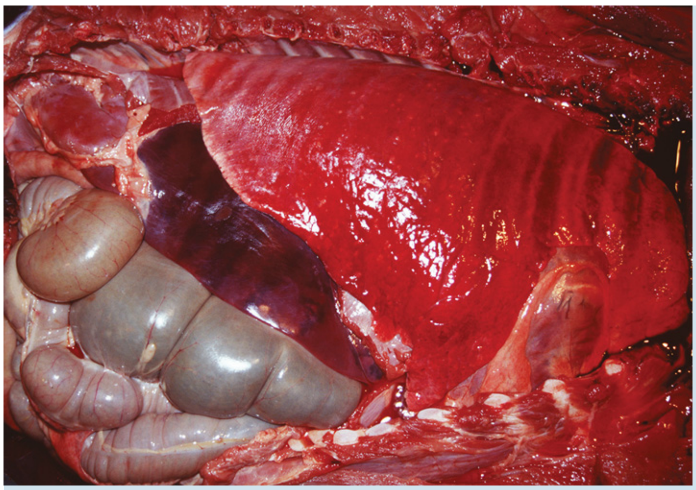


Figure 10.7: Same foal as in Fig 10.6. Note the rib impressions on the lung surface.

should be considered in EHV-1 infected foals. This would include all common bacterial agents, in addition to equine viral arteritis which could mimic EHV-1 infection in the neonatal foal. EHV-1 infected foals commonly have concurrent bacteremia due to immunosuppression.

Diagnosis

- The diagnosis should be suspected if there is known activity of EHV-1 abortion and/or respiratory disease on the farm and a neonatal foal develops the clinical signs listed above.
- Liver enzymes may or may not be elevated more than other foals with just bacterial sepsis, so serum chemistry will not always be helpful.
- Foals with EHV-1 are almost always leukopenic, but this may also be seen with acute bacteremia.
- Buffy coat and bone marrow PCR for EHV-1 may provide an antemortem diagnosis in some cases.
- Confirmation of the disease is usually at necropsy by finding severe diffuse pneumonia, icterus, and hepatic necrosis with viral inclusion bodies.
- On gross examination of the liver, *Actinobacillus* spp and *Streptococcus* spp, which are also common causes of sepsis and acute death in neonatal foals, may cause a similarly abnormal appearing liver. *Actinobacillus* spp usually also has multifocal necrosis of the kidney and absence of diffuse pneumonia.

Treatment

This disease in foals is usually hopeless although on occasion a foal may survive. (See Chapter 5, pp. 147–148.)

Liver failure in foals following neonatal isoerythrolysis (NI) (Fig 10.11)

History

Several foals have developed liver failure following a prolonged or refractory course of NI which required two or more transfusions. The etiology of this liver failure is unknown, but may be a result of chronic hypoxia to the liver and/or iron toxicity associated with multiple transfusions or, more likely, a cholangiopathy associated with the hemolysis and bile stasis.

Figure 10.11: Severe icterus and bilirubinuria (urine in cup) of a foal recovering from neonatal isoerythrolysis requiring two transfusions and then developed liver disease (marked elevation in GGT) and failure.

Clinical signs and diagnosis

- Following prolonged but eventually successful treatment for NI, the foal is noticed to remain lethargic and may be noticeably jaundiced or may not grow as well as other foals the same age.
- A serum biochemistry confirms hepatic failure with elevations in bile acids and direct bilirubin.
- The liver enzymes are all elevated with the GGT elevation most pronounced.
- Ultrasound exam and biopsy of the liver are both abnormal with the ultrasound exam revealing increased echogenicity and irregularity of appearance.
- The biopsy generally is reported as a hepatopathy with both regeneration and fibrosis.

Treatment

There is no proven treatment for this poorly understood liver problem. Certainly drugs that may decrease inflammation and/or oxidative injury in the liver and promote bile flow would be recommended. These include pentoxifylline (8.5 mg/kg PO q8–12 h), S-adenosylmethionine (SAMe) (20 mg PO q24 h), and supplemental vitamin E and selenium.

Bile duct obstruction (Figs 10.12–10.17)

Etiopathology

In the equine, there are two biliary openings into the duodenum – major and minor biliary papillae. Both drain into the most proximal duodenum in very close proximity.

Foals with gastroduodenal ulcers may develop strictures of the duodenum associated with healing of the duodenal ulcer. If the stricture is at the site of the biliary opening, obstruction of the bile flow

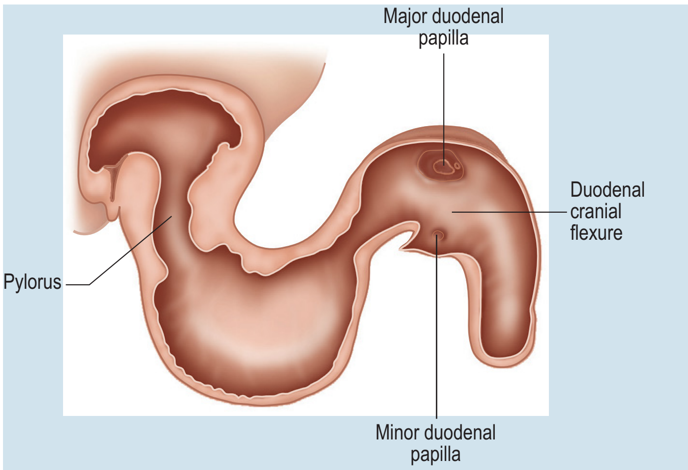


Figure 10.12: Schematic drawing illustrating the locations of the major and minor equine biliary papillae openings.

Figure 10.13: A 6-week-old foal with ptyalism, odontoprisis and diminished nursing. Upon passage of a nasogastric tube, 3 liters of milk reflux was obtained. The foal had diarrhea for several days, 2 weeks prior to presentation.

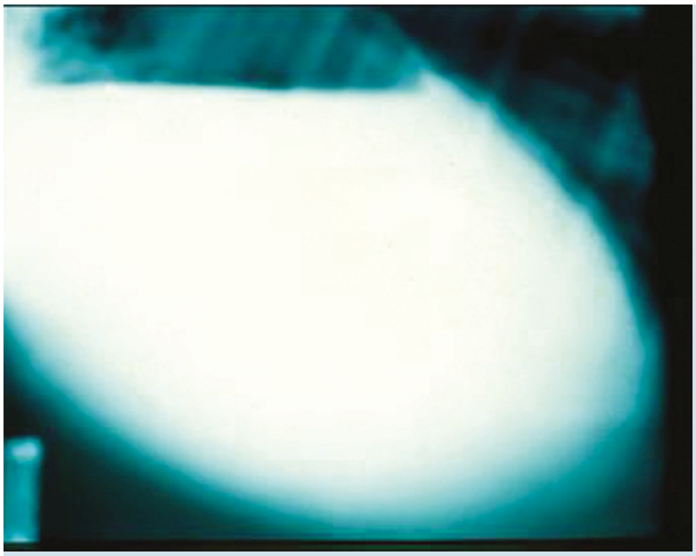


Figure 10.15: Barium study of the foal showing gastric distention and failure of barium to leave stomach.

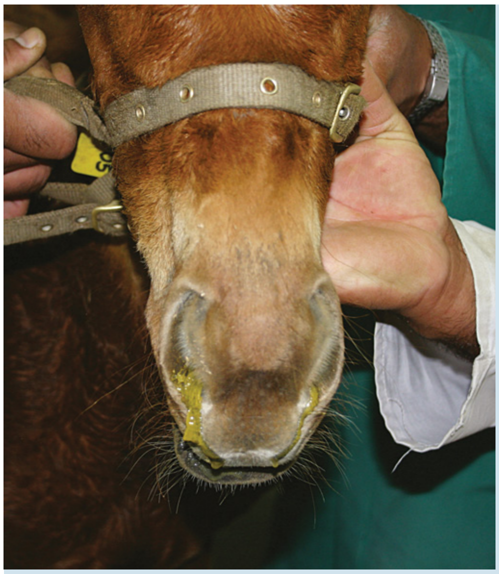


Figure 10.14: Gastric reflux evident at the nostrils of a 6-week-old foal with a duodenal stricture.

Figure 10.16: Barium study of another foal showing barium in the biliary tract confirming duodenal obstruction beyond the opening of the common biliary papillae.

will occur. If the stricture is distal to the opening, there will be reflux of ingesta into the bile ducts. Reflux of ingesta results in a cholangiohepatitis.

The clinical signs are similar with stricture at either site, but the prognosis differs – being grave for strictures at the biliary opening.

History and clinical signs

- There is often a history of a previous illness, e.g. diarrhea, from which the foal seemingly recovered.
- Either during the primary illness or soon thereafter, the foal begins to show clinical signs of prolonged gastric outflow, ptyalism, colic, bruxism and decreased nursing.
- If there is obstruction of the bile, the foal will be icteric.
- Signs are usually in seen in foals at 2–6 months of age but can be seen as young as 2 weeks.

Diagnosis

- The history and clinical signs are characteristic of delayed gastric emptying due to ulceration.
- If a duodenal stricture is present and ultrasound exam or radiographs are taken prior to endoscopy, an enlarged stomach with an obvious fluid line can be seen.
- Endoscopy may reveal esophageal ulcers and gastric ulceration. The stomach may have a large amount of milk present which needs to be refluxed in order to complete the endoscopic exam.
- Duodenoscopy will reveal a stricture of the pylorus, duodenal ampulla or at the duodenal cranial flexure (location of the major and minor duodenal papilla).

- If the stomach is obviously enlarged, a barium study should be performed to help determine the severity of the duodenal obstruction. If the duodenal obstruction is nearly complete and distal to biliary opening, barium may be seen in the bile ducts, but there will be little or no barium leaving the stomach 30 minutes after administration.

Treatment

- Medical treatment should include intravenously administered H_2 blockers, e.g. ranitidine, sucralfate and antacids.
- Other treatments include misoprostil (3–5 µg/kg PO q6–12 h) for its mucosal protective effects and bethanechol (0.025 mg/kg SQ q8 h) as a prokinetic.
- Supportive care such as intravenous fluid administration, regular gastric decompression and parenteral nutrition may be required. The administration of DMSO may help to reduce pyloric edema which is frequently seen in the acute stages. Nothing should be administered per orem while gastric reflux is present.
- If the history, clinical signs, endoscopic and radiographic findings suggest chronic obstruction, then medical therapy is unlikely to be curative.

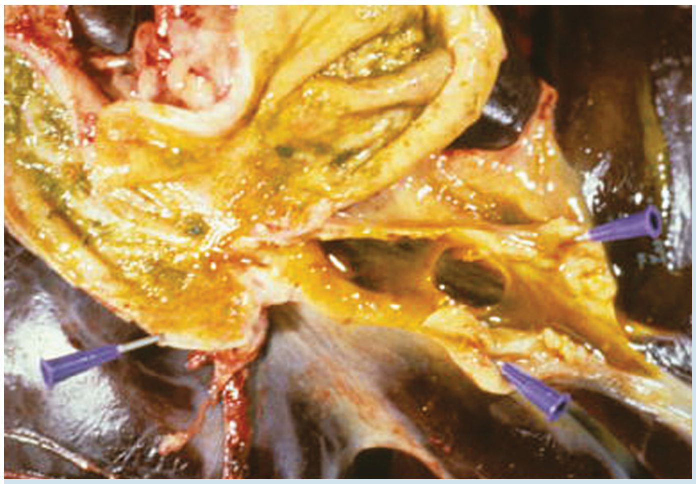


Figure 10.17: Necropsy of foal in Fig 10.16 with bile ducts dissected, demonstrating ascending cholangiohepatits.

Umbilical vein abscess into the liver (Figs 10.18 & 10.19)

Etiology

Foals, particularly those with inadequate colostral antibody absorption, are predisposed to infection of the umbilical structures. In many cases, the umbilical vein may be infected and enlarged (>1 cm) extending from the umbilicus into the liver. A variety of Gram-negative enterics, Gram-positive cocci and occasionally anaerobic bacteria may be cultured from the abscessed vein.

Clinical signs

- Affected foals are usually aged 3–14 days when the diagnosis is made.
- Foals are febrile, depressed, may be lame from infectious arthritis, and have signs of pneumonia.
- The urachal area may or may not be abnormal.
- Complete blood counts reveal leukopenia or leucocytosis frequently accompanied by a left shift.
- Other markers of inflammation (low serum iron, high fibrinogen) are usually present on serum chemistry profile as expected.
- Liver enzymes are usually normal.

Diagnosis

The diagnosis is based upon signalment and clinical signs, and ultrasound examination of the umbilical structures. Occasionally, if

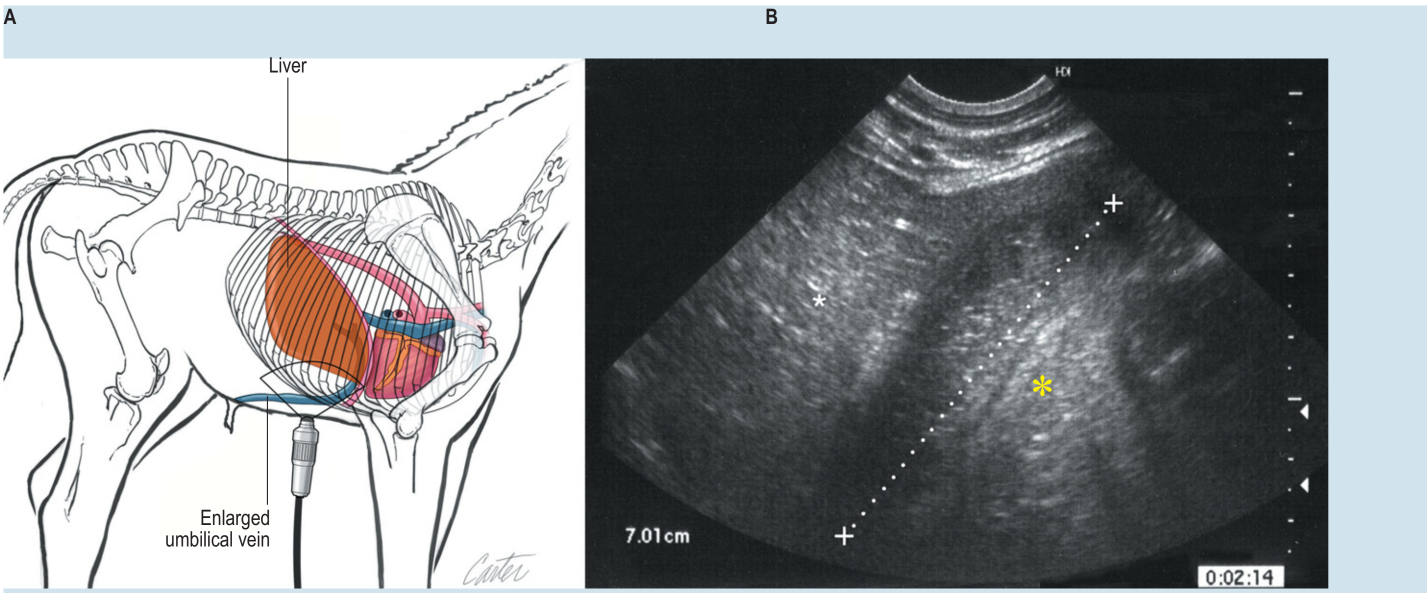


Figure 10.18: Schematic representation and ultrasonographic image of the foal's liver showing the enlarged umbilical vein (dotted line) within the liver parenchyma (*) (from Divers TJ, Perkins G 2003 Urinary and hepatic disorders in neonatal foals. Clinical Techniques in Equine Practice 2:67–78).

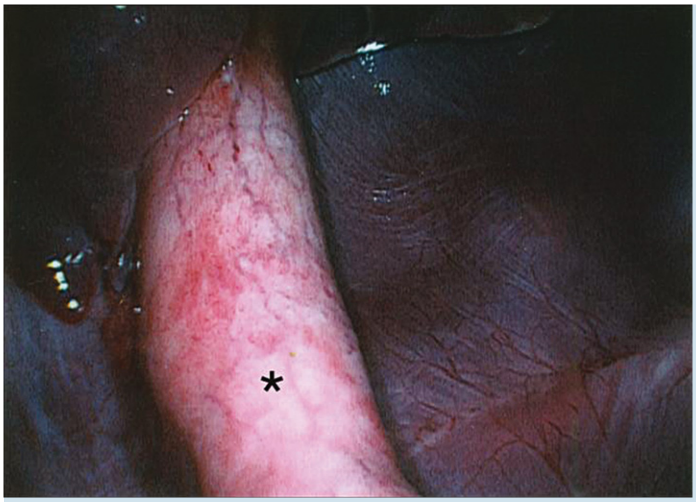


Figure 10.19: Appearance of umbilical vein (*) during laparoscopic surgery.

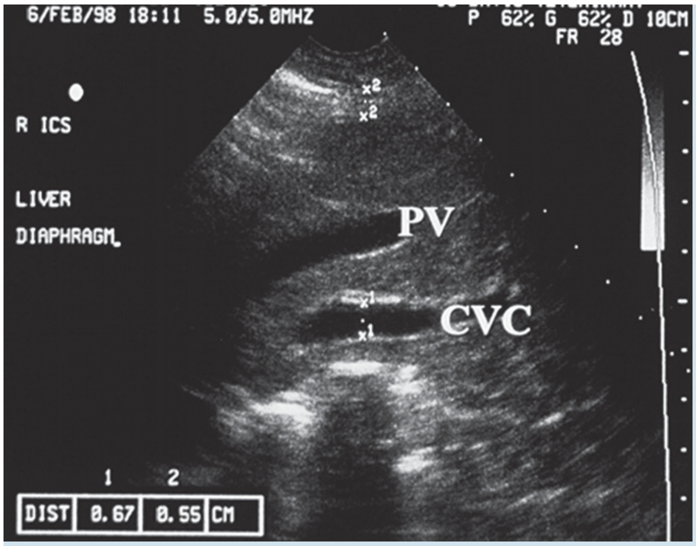


Figure 10.20: Sonogram of a normal liver. Note the portal vein (PV) and the central vena cava (CVC).

the foal can remain relaxed in lateral recumbency, the umbilical vein can be palpated through the abdominal wall.

Treatment

- Treatment depends primarily on the clinical condition of the foal and the severity of the infection. In most instances treatment with broad-spectrum antibiotics (e.g. chloramphenicol or trimethoprim sulfamethazole with rifampin) is initiated.
- Depending on the site of infection it may be possible to obtain a sample for culture. Though blood cultures frequently fail to yield a growth in these cases they are worth doing as a positive result if obtained will be invaluable in guiding treatment.
- The length of antibiotic treatment depends on the response but is prolonged in most cases. The response should be assessed by both the improvement in the clinical condition of the foal and ultrasound appearance of the lesion.
- If the abnormality extends into the liver, surgery is recommended. It may also be indicated in certain foals that present in a serious clinical condition or in foals that have failed to respond appropriately to medical therapy. The vein is either removed as far into the liver as possible and the stump cauterized, or the distal part of the vein is removed and the remaining vein (running into the liver) is marsupialized to allow drainage.
- Plasma is usually administered to all foals with failure of passive transfer.
- Unless there is severe organ dysfunction (pneumonia), or infectious arthritis, the prognosis with appropriate therapy is good.
- If the umbilical vein is marsupialized, this does require significant after-care in some cases.

Portal vein thrombosis (PVT) (Figs 10.20 & 10.21)

This is a rare occurrence in foals secondary to another illness. The thrombosis may be septic, e.g. *R. equi*, or occur due to enteritis, a generalized sepsis and coagulopathy.

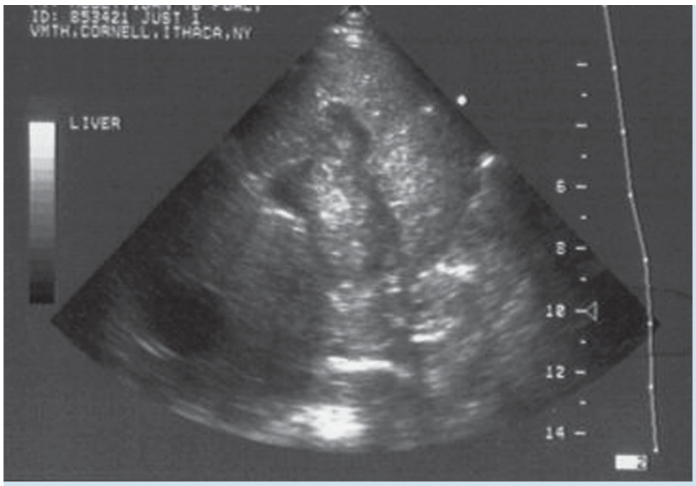


Figure 10.21: A 6-week-old TB filly with persistent fevers, septic arthritis, elevated liver enzymes and a portal vein thrombus. Ultrasonogram from the foal showing a thrombus which was estimated to be occluding 90% of the vein. The foal recovered with long-term antimicrobial therapy.

Clinical signs

- The signs attributed to the PVT include depression, presumably due to hepatic shunting of gut-derived proteins, and diarrhea if the thrombotic occlusion is acute and complete.
- The diarrhea is likely a result of increased mesenteric venous pressures, although in some cases, diarrhea and sepsis might be responsible for the thrombosis.
- If the thrombosis is septic, persistent fevers would be expected.

Diagnosis

The diagnosis is unlikely to be made antemortem unless there is an increase in hepatic derived enzymes and ultrasound examination is performed that demonstrates the thrombus.

Figures 10.22 & 10.23: A 2-month-old foal that had liver failure on day 3 after receiving an iron/probiotic supplement prior to colostrum. The foal seemingly recovered but developed hepatoencephalopathy again (head pressing, bucket chewing) at 2 months.

Treatment

- Most cases are found at necropsy so there is little experience with treatment.
- One foal with nearly 90% occlusion of the portal vein was treated with macrolides and rifampin and recovered.
- Anticoagulant therapy such as aspirin and/or pentoxifylline might be of benefit.

Toxic hepatopathy (Figs 10.22–10.24)

Etiopathology

Although many (several) drugs administered to foals, e.g. NSAIDs, rifampin, antifungal drugs, inhalants, herbal products, anti-ulcer medication, etc. have been temporarily associated with elevations in hepatic enzymes, only iron has been documented to cause toxic hepatic failure. The iron-induced hepatic failure in foals only occurs when iron is administered prior to colostrum.

Figure 10.24: Liver of the foal in Figs 10.22 & 10.23 at necropsy showing attempts at regeneration among areas of severe cirrhosis.

Clinical signs

Typically 3–5-day-old foals with iron-induced acute liver failure have an acute onset of hepatoencephalopathy (coma, blindness, seizure, etc.) which is almost uniformly fatal.

Diagnosis

The diagnosis is based upon history (iron administration prior to colostrum), clinical signs and laboratory findings of hepatic disease and dysfunction.

Treatment

Intensive supportive care with crystalloids, dextrose, branch chain amino acids and "gut sterilization" with neomycin has been attempted, but has rarely been successful. Products with iron should not be given to foals in the first days of life.

Portosystemic shunt (Figs 10.25–10.27)

History

Congenital portosystemic shunts in foals are rare. When present, clinical signs may be noted soon after birth or may not be observed until 2–6 months of age when the foals begin to eat sweet feed and/or roughage. Shunts may be single or multiple, intrahepatic or extrahepatic.

Clinical signs

- The signs are mostly a result of hepatoencephalopathy and include depression, recurrent seizures, ataxia and cortical blindness.
- Signs may wax and wane and affected foals may become unthrifty.

Figure 10.25: Acute onset of ataxia, wandering, and blindness in a 2-month-old Belgian foal with a portosystemic shunt. Blood ammonia was >300 μmol/L.

Differential diagnosis

- Since the predominant signs are abnormal behavior and, in some cases, poor growth, any chronic disease that causes this combination of signs in similarly aged foals is a differential.
- Brain abscess, mesenteric and/or lung abscess, microencephaly, hydrocephalus should be ruled out.
- Morgan foals with hyperammonemia may have very similar clinical signs.

Diagnosis

- The diagnosis may be difficult in some cases since liver enzymes and serum bilirubin are usually normal.
- ***Markedly elevated serum bile acids and blood ammonia with normal serum hepatic enzymes is highly supportive of the diagnosis.***
- The diagnosis can be confirmed by a portogram or by nuclear scintigraphy.

Treatment

Surgical repair has been reported in one foal. Single extrahepatic shunts would have a better prognosis than multiple or intrahepatic shunts.

Hyperammonemia of Morgan foals (Fig 10.28)

Etiology

This is presumed to occur because of an inherited abnormality in hepatic ammonia metabolism.

Signalment and clinical signs

Affected Morgan foals are usually 4–7 months old and have an acute onset of signs caused by cerebral dysfunction. These signs include blindness, head pressing, circling and seizures. In some cases, there is hemoglobinuria (red discoloration of the urine).

Figure 10.26: Technetium being administered rectally to a foal with portosystemic shunt.

Diagnosis

- The diagnosis is based upon signalment and clinical signs in addition to measurement of blood ammonia and other hepatic enzymes.
- Blood ammonia levels are often very high (300–600 μmol/L) in the face of normal readings for other hepatic enzymes. If blood ammonia is to be measured, it should be performed either "in house" with a control sample or, if it is sent to a laboratory, serum should be immediately collected and frozen and a control sample handled in the identical way.
- Liver histopathology is normal.

Treatment

- Although the disease has been uniformly fatal, some cases will improve for several days only to have a second onset of severe neurological signs later.
- Treatments could include mannitol (0.5–1.0 mg/kg IV q12 h), neomycin (4–8 mg/kg PO q8 h) or metronidazole (10–15 mg/kg PO q8–12 h) and lactulose (50–100 mL PO or PR q6–12 h), in addition to intravenous crystalloids supplemented with potassium chloride.
- If the foals have maniacal behavior, lower end dosages of an alpha$_2$ agonist can be used to mildly sedate the foal.

Hepatic lipidosis (Fig 10.29)

Etiology

This may occur due to insufficient caloric intake and/or increased breakdown of adipose tissue in miniature foals. This metabolic condition (hyperlipemia and hepatic lipidosis) is rare in neonatal

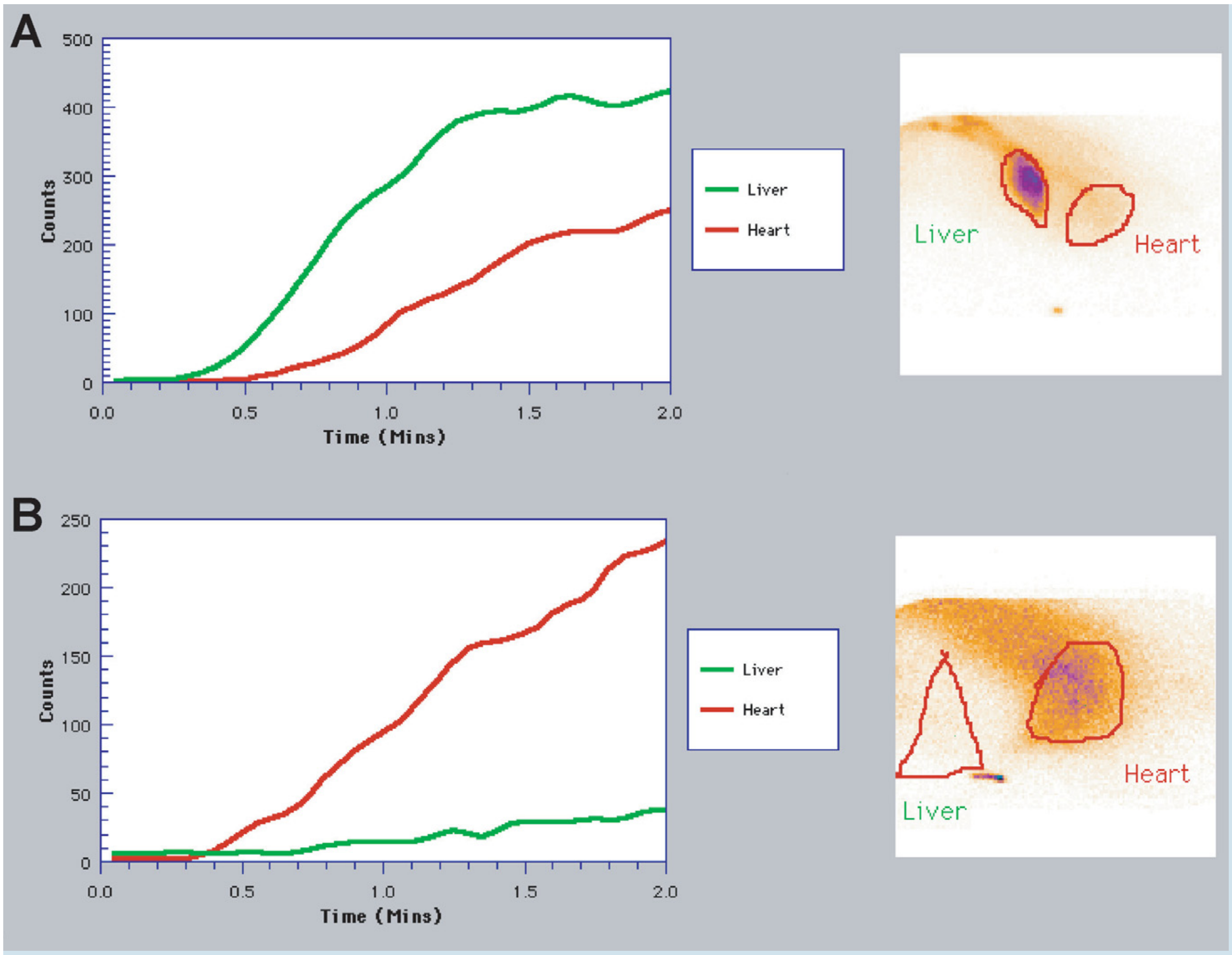


Figure 10.27: Time activity curves (left) and composite images (right) are derived from transcolonic portal scintigraphy of two foals. The procedure was performed using 30–35 mC of Technetium pertechnetate per rectum, A dynamic study was performed acquiring 40 frames over 2 minutes, Regions of interest were drawn over the areas of the liver and heart using the composite image. The activity in these regions was plotted as counts per unit time to produce a time–activity curve. (a) Normal foal with activity detected in the liver before the heart, (b) Foal with portosystemic shunt with activity detected primarily in the heart and minimal activity in the liver (from Divers TJ, Perkins G 2003 Urinary and hepatic disorders in neonatal foals. Clinical Techniques in Equine Practice 2:67–78)

foals other than miniature foals. Hepatic lipidosis may also rarely occur due to prolonged administration of high levels of corticosteroids or inappropriately formulated TPN solutions.

Clinical signs

The clinical signs are generally:

- anorexia (which may also occur due to a primary or triggering disease)
- ventral edema
- cerebral cortical signs (blindness, depression)
- shock and acute death may occur due to rupture of the liver.

Diagnosis

The diagnosis is based upon:

- signalment (miniature equine)
- history of a predisposing disease or event
- biochemical findings of elevated plasma triglycerides and liver enzymes
- the plasma may be a white color due to the marked elevation in triglycerides (hyperlipemia).

Treatment

- Treatment should be directed towards any predisposing disease and nutritional support for the hepatic lipidosis/hyperlipemia.
- Parenteral nutrition with amino acids and glucose often combined with insulin can be life saving in some cases.
- Enteral support via nasogastric tube should be provided. If possible, a low fat milk replacer or mare's milk mixed with yogurt and/or glucose and galactose is used.
- Crystalloids should be given intravenously to maintain hydration and prevent azotemia. Multi-B vitamins given slowly IV are also recommended.

Figure 10.28: A 4-month-old Morgan with hepatoencephalopathy and hyperammonemia but with only mild to moderate evidence of hepatic disease on histopathology.

Figure 10.29: Antemortem rupture of liver in a miniature donkey foal with hyperlipemia and hepatic lipidosis.

Peritoneal disorders

Peritonitis (Figs 10.30–10.40)

Etiopathology

Peritonitis may result from septic causes (either acute or chronic) or from chemical causes. Acute septic peritonitis in foals is usually a result of intestinal rupture (i.e. gastric or duodenal perforation) or secondary to intestinal inflammation, e.g. Clostridial diarrhea and/or ischemia. Chronic septic peritonitis is usually caused by *Rhodococcus*

Figure 10.30: A 12-week-old foal found collapsed in a paddock with marked abdominal distension. There was acute peritonitis associated with duodenal rupture.

Figure 10.31: The accumulation of fluid within the abdomen secondary to peritonitis has resulted in fluid accumulation within the scrotum. This should be differentiated from uroperitoneum or a urethral tear (see Fig 6.33).

equi or *Streptococcus equi* abdominal abscessation. Chemical/mechanical peritonitis may occur secondary to uroperitoneum, hemoabdomen, parasite migration or surgical handling of the bowel.

Clinical signs

- Signs of acute septic peritonitis include depression, abdominal distension, signs of shock and colic.
- Clinical signs of chronic peritonitis in foals include weight loss, poor hair coat, diarrhea, colic, and "pot belly" appearance. There may also be evidence of multi-organ disease, swollen joints, uveitis, and pneumonia.

Diagnosis

- The tentative diagnosis of peritonitis is based upon history and clinical findings and ultrasonographic examination of the abdomen.
- Ultrasonographic exam will usually show an increased amount of peritoneal fluid that may or may not have increased echogenicity.
- An abscess in the mesentery may be found in some chronic cases.

Figure 10.32: Ultrasonographic image of the abdomen demonstrating fluid accumulation associated with peritonitis. In many cases (not in this image) large accumulations of fibrin can be seen adhering to the visceral surface of abdominal organs.

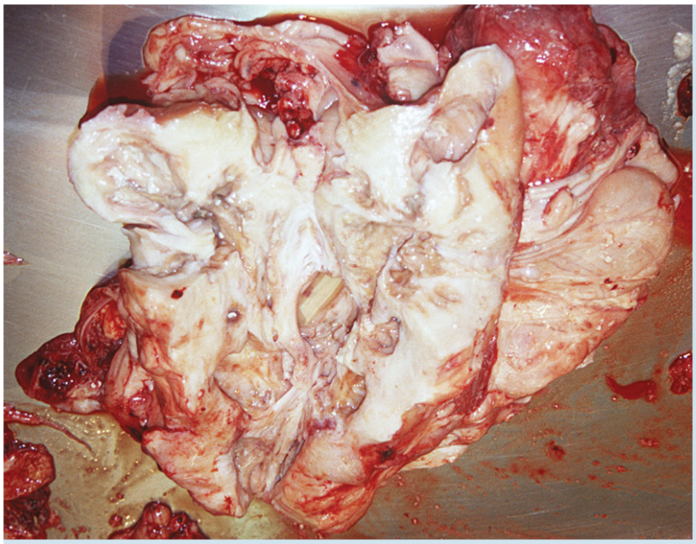


Figure 10.34: *R. equi* mesenteric abscess at necropsy.

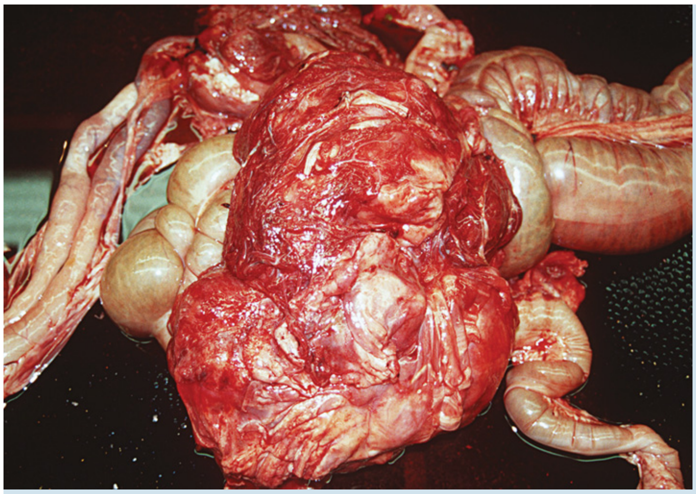
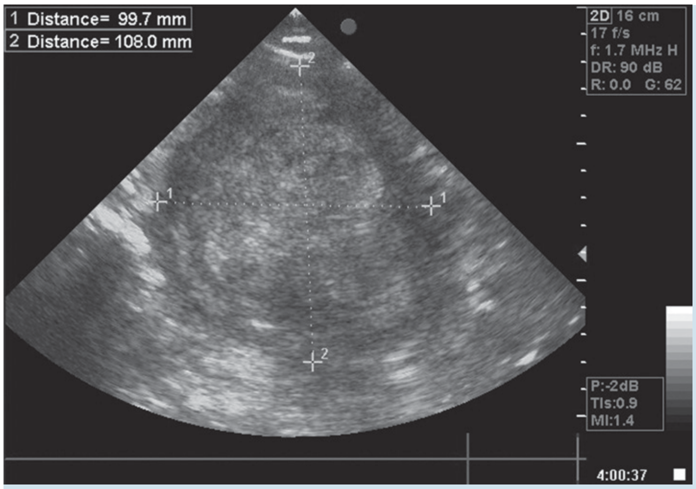


Figure 10.33: Mesenteric *Crossiella equi* (nocardioform) abscess.



Figures 10.35: Ultrasonographic image of the abdomen demonstrating a mesenteric abscess secondary to *R. equi* infection.

- Abdominocentesis should be performed to confirm the diagnosis and look for a causative organism. The white cell count and protein will be increased in the peritoneal fluid and bacteria may be observed or cultured, depending upon how "walled off" the abscess is (for method and interpretation, see Chapter 4, pp. 79–80).
- With acute septic peritonitis such as seen with *Clostridium perfringens* type C, bacteria are usually seen on cytological exam. If the peritonitis is due to intestinal leakage, a mixed bacterial population may be observed.

Treatment

- Treatment of both acute and chronic peritonitis is often unrewarding.
- With acute peritonitis, surgical attempts to repair or remove the affected bowel, in addition to medical support (fluids and antibiotics), have been largely unsuccessful.
- Primary septic peritonitis in which bacteria are sequestered into the cavity from the blood has been treated successfully with abdominal lavage and parenteral antibiotics.
- Chronic peritonitis secondary to abdominal abscess(es) is usually treated with appropriate antibiotics based upon etiologic agent. Surgical drainage can be considered depending upon the size and number of abscess(es).

Omental prolapse (Fig 10.41)

Cause

Following abdominocentesis (usually with a teat cannula), foals with increased abdominal pressure/distension may prolapse the omentum through the centesis site.

Figure 10.36: Same foal as in Fig 10.30. Note the large amount of discolored abdominal fluid obtained by abdominocentesis. Cytological examination of this fluid revealed the presence of feed material.

Clinical signs and diagnosis

A piece of tissue is noted to be protruding from the site of the peritoneal tap.

Treatment

Any disorder causing abdominal distension should be treated appropriately. The protruding omentum should be severed at the body wall and a bandage applied around the abdomen. Application of a small amount of antibiotic ointment to the bandage where it overlies the omentum prevents the omentum adhering to the bandage, which would result in further prolapse on bandage removal.

Chyloabdomen (Fig 10.42)

History

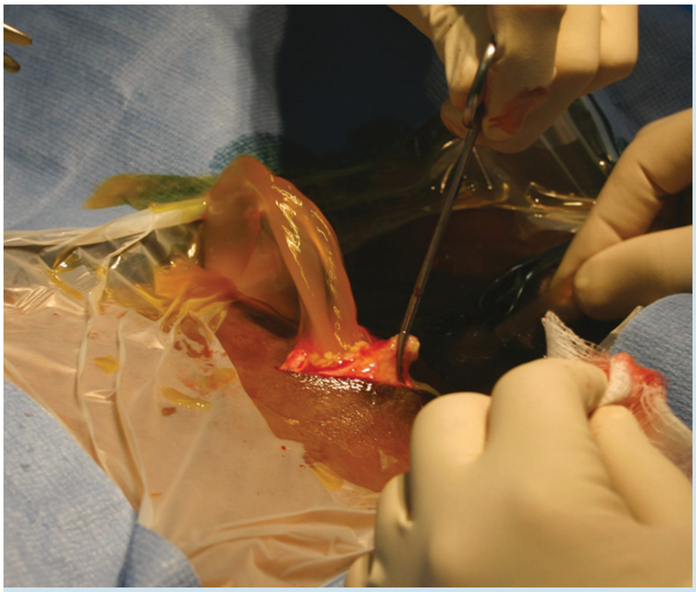
This is a rare condition in foals that is not well understood but is likely related to congenital or acquired obstruction of lymph vessels.

Clinical signs and diagnosis

Cause and effect are unproven but affected foals usually have signs of colic. The diagnosis is made by performing a peritoneal tap and finding white/milky peritoneal fluid.

Treatment

- If the colic is mild and ultrasonography does not reveal any severe abnormality (severely thickened bowel, ileus, enlarged lymph nodes), supportive medical treatments with fluids and analgesics may result in recovery.
- If colic is persistent and there is thickened intestinal wall and enlarged lymph nodes on ultrasound examination, exploratory surgery would be indicated.
- If there is a segment of abnormally thick intestine with dilated lymphatics and white plaques in the same area, resection of the affected intestine and mesentery is recommended.



Figures 10.37 & 10.38: Ultrasound examination and peritoneal fluid analysis of this 7-week-old foal indicated peritonitis but did not indicate a cause (no evidence of bacteria or feed material on cytological examination). An exploratory celiotomy was performed to determine if a bowel rupture was present. Note the copious amount of peritoneal fluid in Fig 10.37 and the hyperemia of the bowel and associated fibrin in Fig 10.38. The peritonitis in this case was primary septic and treatment was unsuccessful.

Splenic disorders (Figs 10.43 & 10.44)

Disorders of the spleen are rare in foals. Trauma resulting in splenic hematomas or hemoabdomen can occur. Most commonly these are

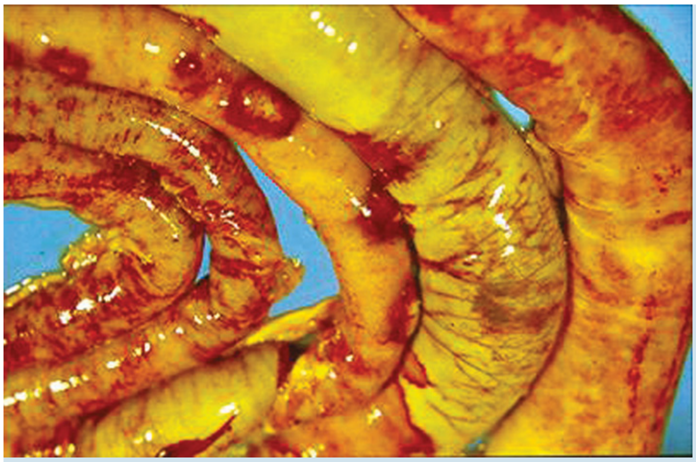


Figure 10.39: Intestine of a 2-day-old foal that died from ***Clostridium perfringens*** type C and had septic peritonitis. Large numbers of Gram-positive rods were present antemortem in the peritoneal fluid.

Figure 10.40: Treatment of an abdominal abscess by draining and flushing. Note the Foley catheters that have been placed in the abscess.

Figure 10.41: Omentum protruding from the site of a peritoneal tap (teat cannula was used) that had been performed in a foal with uroperitoneum. The protruding omentum was cut and removed and the abdomen bandaged.

Figure 10.42: Surgically removed intestine and mesentery from a foal with mild colic and chyloabdomen. Lymphatics are prominent. ***Streptococcus*** was cultured from the mesentery.

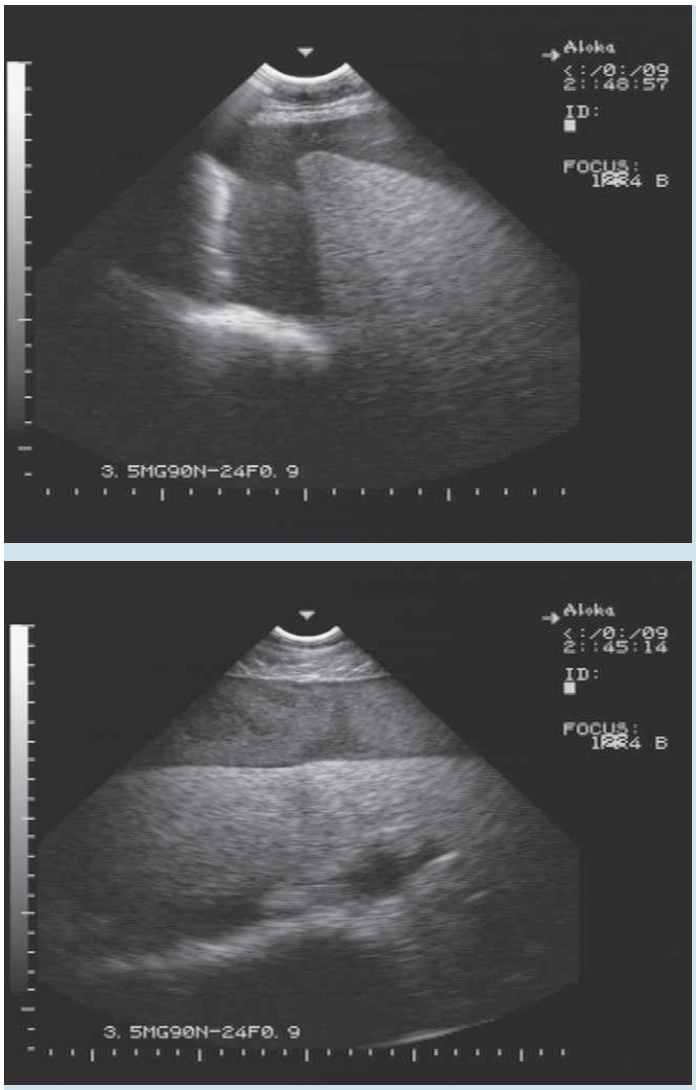


Figure 10.43: Ultrasonograms of a hemoabdomen associated with a splenic tear. These images are from an adult, note the swirling "smoke-like" appearance of the blood.

associated with caudal rib fractures. Severe trauma with large tears usually results in rapid death and the history may simply consist of a foal being found dead in a paddock.

Recommended reading

Divers T1997 Tyzzer's disease. In: Robinson NE (ed) Current therapy in equine medicine IV. Saunders, Philadelphia, p 218–219

Figure 10.44: Basal splenic tear on the visceral surface of a 45-day-old foal. The cause of the tear was unknown with no evidence or history of trauma. A splenectomy was performed and the foal did well.

Fortier LA 2002 Hepatic diseases in foals. In: Mair T et al (eds) Manual of gastroenterology. Saunders, Philadelphia, p 513–526

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CHAPTER 11

The immune system

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Introduction

Foals are born immunocompetent, that is they are able to mount an immune response to a specific pathogen; however it takes 10–14 days for this to become protective. It is during the period between exposure to the pathogen and the development of the protective immunity that the neonate is vulnerable to infectious agents. Therefore it is essential that they receive passive immunity from the dam, to protect them in the first month of life. There is no transplacental transfer of immunoglobulins to the equine fetus; therefore the result is a neonate which is hypogammaglobulinemic and immunonaive. The foal depends on the ingestion and absorption of immunoglobulins in colostrum for passive immunity.

Immunodeficiencies

- Immunodeficiencies arise from the absence or failure of one or more aspects of the immune system.
- They are broadly categorized as primary or secondary.
  - Primary disorders are those in which there is a defect in the maturation of the lymphocytic cells of the immune system; these are either proven or presumed genetic in origin.
  - Secondary immunodeficiencies are those which are acquired or are the result of external or environmental conditions. Failure of passive transfer is the most commonly encountered immunodeficiency in horses.

Primary immunodeficiencies

Severe combined immunodeficiency (SCID) (Fig 11.1)

This is a fatal disease described in Arabian foals, man, dogs and mice. There has also been a reported case in an Appaloosa foal. In the Arabian population the disease is inherited as an autosomal recessive trait. This means that if two carriers or heterozygous animals are mated, 25% of the progeny will be homozygous or affected, 50% will be normal but carriers, and 25% will be normal non-carriers.

Affected foals lack functional B and T lymphocytes, which means that they are unable to mount either humoral or cell-mediated immune responses. Affected foals lack the activity of the enzyme deoxyribonucleic acid-dependent protein kinase (DNA-PK). This enzyme is responsible for the differentiation and normal function of B lymphocytes and T lymphocytes.

History

Affected foals are normal at birth, with the onset of clinical signs determined by the extent of maternal antibody transfer and the rate at which these antibodies are eliminated from the foal's system.

Clinical signs

- Onset of symptoms usually occurs between 2 weeks and 2 months of age.

Figure 11.1: Arabian foal with SCID; note the serous nasal discharge.

- Lack of B and T lymphocytes makes the affected foals unable to fight infections once maternal immunity has faded. Even with adequate maternal antibodies, these foals are still susceptible to intracellular bacterial and protozoal infections due to the lack of T cell responses.
- Initial infections commonly occur in the respiratory and gastrointestinal tracts.
- Adenovirus is a significant pathogen in SCID foals. Other pathogens associated with SCID include *Pneumocystis carinii*, *Cryptosporidium parvum* and *Rhodococcus equi*.
- Affected foals rarely survive beyond 5 months of age.

Diagnosis

- Presumptive diagnosis can be made on signalment, history, clinical signs, complete blood count (CBC) and immunoglobulin analysis.
- CBC shows a lymphopenia (<1000 lymphocytes/μL blood).
- Quantitative immunoglobulin analysis on presuckle serum will show a lack of IgM.
- Definitive diagnosis is now possible using DNA testing. This allows for the premortem diagnosis of affected foals (homozygous) and also the detection of carriers (heterozygous). Carrier stallions are still used for breeding but owners are advised to have their mares tested prior to breeding to such a horse.

Treatment

- No specific treatment or medical management is recommended in SCID foals.
- There is one report of a 32-day-old affected foal that received a bone marrow transplant from a full sibling and survived until 5 years of age, at which time it died due to an unrelated disorder.

Prognosis

- Once definitively diagnosed euthanasia is recommended.
- The aim is to eradicate SCID, by removing all affected animals from the stock. Selective breeding of carriers to non-carriers in order to preserve other desirable traits is carried out.

Agammaglobulinemia

This immunodeficiency is described in Standardbred, Thoroughbred and Quarterhorse foals and is characterized by the absence of circulating B-cells and hence the lack of production of immunoglobulin and specific antibodies. The lack of B-cell function results in an absent or poor response to immunization while T-cell numbers and function are normal. All reports are in male animals, which suggests an X-linked mode of inheritance, similar to X-linked agammaglobulinemia described in humans.

Clinical signs

- Onset of signs coincides with the waning of maternal antibodies between 2 and 6 months of age.
- Persistent infections, most commonly of the respiratory tract and of joints, have been described. Dermatitis, enteritis and laminitis can also occur.

Diagnosis

- These foals will have normal peripheral lymphocyte counts, due to normal numbers of T lymphocytes, but no detectable B lymphocytes.
- Quantitative immunoglobulin analysis will show no detectable IgM or IgA and very low or absent levels of IgG in their serum.
- As yet there is no available genetic test to determine affected or carrier status of foals.

Treatment

Once diagnosed, treatment is not recommended and parents of the affected foal should not be bred from. However, many associated infections may appear to temporarily respond to antibiotics and intravenous plasma.

Prognosis

- The prognosis for agammaglobulinemic foals is poor, with reported survival to 18 months of age. This much longer life span than reported in SCID foals has been attributed to the normal T lymphocyte population and function.

Transient hypogammaglobulinemia

With this immunodeficiency the onset of immunoglobulin production is delayed. In a normal foal immunoglobulin production starts before birth with detectable IgM at 190 days of gestation; however in these foals production may not start until 3 months of age.

This disorder has been reported in Arabian and Thoroughbred foals. A genetic basis is suspected but unproven as yet. The exact

pathogenesis is unknown but similarities have been drawn to transient hypogammaglobulinemia seen in humans.

Clinical signs

Recurring infections start once maternal antibodies wane. These foals tend to be smaller and less developed than non-affected foals of the same age.

Diagnosis

- Complete blood count shows normal circulating lymphocytes, with normal B and T cell numbers.
- Quantitative immunoglobulin analysis shows significantly reduced IgG with IgG (T), IgA and IgM below or within the low to normal range.
- It is possible that this disorder is under diagnosed due to its transient nature.

Differential diagnosis

Due to the excellent prognosis in these cases it is important to differentiate transient hypogammaglobulinemia from agammaglobulinemia. The latter exhibits lack of detectable IgM and IgA with very low or absent IgG and a circulating B cell deficiency, in contrast to transient hypogammaglobulinemia which shows the presence of circulating B cells, and reduced but detectable immunoglobulin.

Treatment

- Once diagnosed the foal should be placed in an isolated area to avoid contact with other animals, which may introduce either opportunistic or pathogenic organisms.
- Supportive care consisting of antibiotics and exogenous antibodies may be necessary to treat recurring infections.
- Routine quantitative immunoglobulin analysis should be carried out to assess onset and progression of immunoglobulin production.

Prognosis

The prognosis for animals suffering from transient hypogammaglobulinemia is favorable once they receive the appropriate supportive care.

Selective IgM deficiency

This is seen most commonly in Arabians and quarter horses but has been described in other breeds. Affected horses are of both genders and age of onset varies from 2 months to 5 years of age. IgM levels in all affected animals are non-detectable or severely decreased. A genetic basis is unproven as yet.

Clinical signs

There are two clinical syndromes seen with selective IgM deficiency:

- The most common affects foals or weanlings less than 1 year of age. They show typical signs of immunodeficiencies including persistent or recurring infections. Antimicrobial therapy may produce an initial but short-lived improvement.
- The second syndrome is diagnosed in horses 2–5 years of age. They have a long history of recurrent infections. Some of the horses in this group will have gross or histological evidence of lymphosarcoma. The exact pathogenesis of this syndrome is not clear.

Diagnosis

- Quantitative immunoglobulin analysis will demonstrate selective IgM deficiency.
- There may be normal or elevated levels of IgG and IgA.
- Peripheral lymphocyte counts will be normal.
- Adult horses with selective IgM deficiency should have further investigation to assess if lymphoreticular neoplasia is present.

Differential diagnosis

On the basis of clinical signs the other primary immunodeficiencies may be included as differentials; however selective IgM deficiency with normal lymphocyte counts will help to rule these out.

Treatment

Supportive treatment with antibiotics and plasma can be given. The half-life of transfused IgM is short therefore treatment is only providing a temporary relief.

Prognosis

- The prognosis for survival is poor with most of the animals suffering from the first syndrome dying before 1 year of age.
- There have been reported cases of survival where foals have developed normal IgM concentrations after several months of supportive treatment. The reason for this is not clear. It is postulated that in these scenarios the IgM deficiency may be secondary to some underlying primary disease.
- In the second syndrome the prognosis is poor in the adults with evidence of neoplasia.

Fell pony syndrome (anemia, immunodeficiency and peripheral ganglionopathy) (Figs 11.2–11.4)

FPS is a recently described syndrome of immunodeficiency and anemia of young Fell pony foals. There is no sex predilection. Affected foals are normal at birth with onset of clinical signs occurring at 2–4 weeks of age.

Clinical signs

- Affected foals lose condition and become weak and lethargic.
- Specific signs associated with FPS include diarrhea, coughing, hypersalivation and chewing motions.
- Failure to respond to therapy with progressive anemia and lethargy is a common sequel leading to death or euthanasia.

Diagnosis

- A presumptive diagnosis can be made on the presence of clinical signs in a young Fell pony.
- Recent work has identified a significant reduction in immunoglobulins M, G(a), G(b), G(T) and B lymphocytes in affected foals in comparison to non-affected age-matched Fell pony foals.
- Affected foals also are profoundly anemic (PCV < 20%). This anemia is unrelated to haemolysis or blood loss.
- Opportunistic infections include adenoviral bronchopneumonia and cryptosporidial enteritis.

Figure 11.2: Oral candidiasis in a Fell pony (from Knottenbelt DC, Holdstock N, Madigan JE 2005 Equine neonatology, medicine and surgery. Saunders, Philadelphia).

- Postmortem lesions include erythroid hypoplasia of the bone marrow, thymic hypoplasia, lymphoid depletion of the other lymphoid organs, bronchopneumonia, enteritis and glossal hyperkeratosis.
- Peripheral ganglionopathy has been recorded in some cases.

Differential diagnosis

Other immunodeficiencies involving reduction in circulating immunoglobulins and B lymphocytes could be considered in the differentials. Anemia has not been reported in any primary immunodeficiency other than FPS.

Treatment

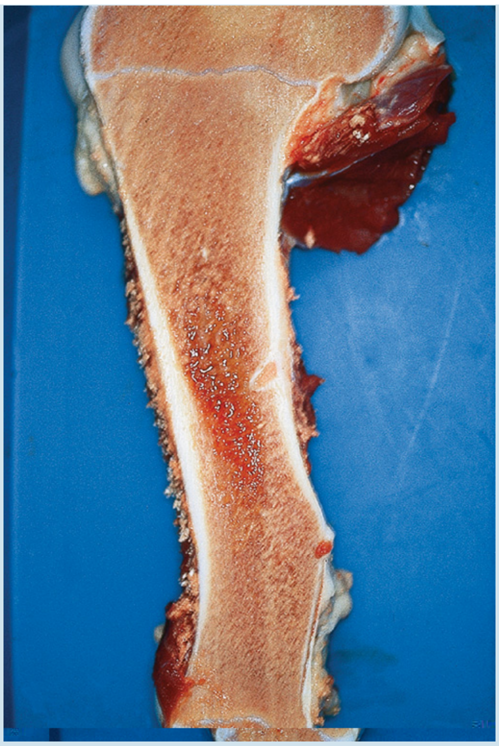
Supportive therapy is not successful with recurrence of clinical signs. Affected foals die or are euthanized before 3 months of age.

Prognosis

This is a congenital fatal disease; further investigations are being carried out to determine if there is an inheritance pattern. The exact etiology of this disease remains unknown.

Miscellaneous primary immunodeficiencies

Selective immunoglobulin deficiency has been described in a 10-month-old Arabian filly foal, where IgG concentrations remained normal but there were low to absent levels of IgM, IgA and IgG (T).



Figures 11.3 & 11.4: Anemia. Pale oral mucous membranes and an almost non-functional bone marrow (from Knottenbelt DC, Holdstock N, Madigan JE 2005 Equine neonatology, medicine and surgery. Saunders, Philadelphia).

Secondary immunodeficiencies

Failure of passive transfer (FPT)

FPT occurs when a foal fails to ingest or absorb adequate colostral immunoglobulins. *This is the most commonly encountered immunodeficiency in foals.*

Normal foals will suckle within the first 3 hours of life and maternal immunoglobulins can be detected in their serum from 6 hours onwards. Passive transfer is assessed by measuring the serum concentration of IgG. There are several tests available to measure serum IgG including radial immunodiffusion, zinc turbidity test, glutaraldehyde clot test, latex agglutination and ELISA based testing (see Ch. 3 p. 59). The ideal IgG level is ≥8 g/L (800 mg/dL) at 18–24 hours.

FPT is a well-recognized risk factor for sepsis; however while the majority of foals that present for sepsis have FPT not all foals with FPT develop sepsis. Foals with complete FPT are at highest risk even with very low environmental challenge.

Neonates with partial FPT are not always at increased risk of sepsis. This would suggest that factors other than immunoglobulin status are responsible for neonatal sepsis. These factors include peripartum and environmental hygiene, pathogen challenge and virulence, and other diseases and stress in the neonate.

Factors that lead to high risk of FPT include:

- *Inadequate volume or loss of colostrum*. Premature lactation is a common cause of the loss of colostrum. Mares that drip or run milk prior to parturition have been shown to have lower colostral IgG levels. Factors that may lead to this include placentitis, premature placental separation and twinning (see Fig 3.9).
- *Poor immunoglobulin concentration in colostrum*. Mares greater than 15 years of age, Standardbred mares and mares that foal early in the year are most at risk of producing colostrum that is low in immunoglobulin content. Colostral immunoglobulin levels can be measured in the field using a colostrometer or sugar (Brix) refractometer (see Chapter 3, p. 58).
- *Failure to ingest colostrum*. Weak, hypoxic or premature foals may not be able to suckle the mare. Death of the dam or rejection of the neonate are other reasons for inadequate ingestion of colostrum.
- *Failure of absorption*. This occurs when foals are known to have ingested adequate quantities of good colostrum and still have FPT. Mostly seen in premature or sick neonates, exact reasons for this are unclear. Suggestions that these foals may catabolically eliminate IgG more rapidly have been made. Also, endogenous or exogenous glucocorticoids may lead to more rapid maturation of the specialized enterocytes – which in turn leads to the decrease in absorption of the immunoglobulins.

History

Any foal where there is a history of any of the risk factors mentioned above is a candidate for FPT.

Clinical signs

- Clinical presentations of foals with FPT are variable and can be attributed to either the initial illness that prevented them ingesting enough colostrum.
- These may include prematurity, dysmaturity, weakness, ischemic encephalopathy, septicemia, septic arthritis, pneumonia, enteritis and other infections.

Diagnosis

- History and clinical signs of concurrent illness may aid diagnosis, but definitive diagnosis can be made by assessing the neonate's IgG status
- Quantitative analysis of IgG is easily and rapidly carried out in the field using one of the many available commercial kits. This allows for the early evaluation of serum IgG levels and when necessary supplementation with immunoglobulins can be carried out.
- Serum IgG concentrations in foals can be determined as early as 6 hours postpartum.
- Any foals that are considered high risk should have their IgG levels measured at 12 hours of age, to allow for early supplementation. Routine evaluation of the normal foal can be carried out at 12–24 hours of age.
- Complete FPT has been defined as IgG <2 g/L and partial failure as 2–8 g/L. It has been suggested that an IgG of >4 g/L is adequate for a healthy foal. However most clinicians in the field aim for an IgG of >8 g/L.

Prevention and treatment

- Once partial or complete failure of passive transfer has been identified, it is universally accepted that foals with IgG <4 g/L should have supplementation. There are differing opinions regarding supplementation in foals with IgG 4–8 g/L.
- All risk factors including concurrent illness, farm history and environmental challenges should be taken into account.
- It may be prudent to supplement any high risk or ill foals regardless of IgG concentrations.
- If identified in a foal less than 12 hours of age, the oral route can be used to supplement IgG as it is likely that absorption will occur.
- Good quality colostrum (specific gravity >1.065, sugar refractometry >20%, or IgG concentration >70 g/L as determined by SRID) that is fresh or has been frozen can be used.
- Donor colostrum should be free from anti red blood cell antibodies to prevent neonatal isoerythrolysis.
- Frozen colostrum should be thawed at room temperature or using a water bath no warmer than body temperature as higher temperatures may denature the immunoglobulins.
- ***A 50 kg foal should receive 0.5–2 liters of colostrum (depending on colostrum quality) this can be given as 500 mL feeds every 1–2 hours.***
- The colostrum can be bottled if the foal will accept this or administered by stomach tube.
- Bovine colostrum has been used if no equine colostrum is available, however the immunoglobulins have a shorter half-life and antibodies may not be specific to equine pathogens.
- Equine plasma can be given orally as the immunoglobulins will be absorbed similarly; however, the concentration is lower than that of colostrum and larger volumes may have to be administered.
- Commercially available concentrated equine IgG products have also been used however their efficacy is questionable and they should not be used as a routine replacement for equine colostrum or plasma.

- Once the foal is over 12 hours of age it is best to parenterally supplement immunoglobulins.
- Plasma from a donor on the same farm as the recipient foal has the advantage of having antibodies specific to pathogens in that environment. However these donors should be screened for infectious agents, be free of anti red blood cell antigens and ideally be Aa and Qa negative. IgG concentration can be determined and should be >12 g/L.
- There are many commercial sources of plasma. The donors in these commercial operations are all screened for infectious agents, alloantibodies and alloantigens that may cause problems. The donors can also be hyperimmunized with specific pathogens or endotoxin to increase the amount of specific antibodies. The plasma has a known IgG concentration.
- The exact amount of plasma necessary to raise the recipient's IgG levels depends on the size of the foal, degree of deficiency and the IgG content of the plasma.
- The presence of sepsis can alter the distribution and catabolism of the IgG, and therefore requires greater volumes of plasma to raise the IgG concentration of the recipient. As a rule of thumb 20 mL/kg of plasma with an IgG concentration of 12 g/L will raise the recipient's IgG concentration by 2 g/L. Most foals that have partial FPT will require 1–2 liters of plasma; however, it may be necessary to administer 2–4 liters of plasma to a foal that has complete failure of passive transfer.
- To assess accurately the IgG concentration after parenteral supplementation a sample should be taken 24 hours post-transfusion; this takes into account the redistribution of IgG to extracellular spaces.
- Plasma should be thawed at room temperature and raised to body temperature before administration. An aseptically placed IV catheter and an in-line blood filter should be used.
- There is very little information available on plasma transfusion reactions, but tachycardia, tachypnoea and muscle fasciculations have been observed. These usually disappear once the rate of flow is reduced (see p. 303).
- Other severe reactions like collapse will necessitate ceasing the transfusion, and initiation of supportive therapy to maintain circulation.
- Appropriate prophylactic antibiotic therapy is indicated in high-risk and FPT foals to reduce the incidence of septicemia.
- Concurrent illness will require specific treatment regimes.

Prognosis

- However multifactorial the development of sepsis, FPT is one of the high risk components that can be prevented.
- Identification of high risk foals, early monitoring of passive transfer, and supplementation with IgG to aim for a serum IgG concentration of >8 g/L at 24 hours of age will help to reduce the incidence of FPT.
- Foaling and environmental hygiene also plays a very important role in the prevention of sepsis. Once sepsis has developed, early diagnosis and aggressive therapy are necessary for a successful outcome.

Perinatal herpes virus infection

See Chapters 5 (p. 147) and 10 (p. 279).

Miscellaneous secondary immunodeficiencies

A single report in the literature describes immunodeficiency associated with oral candidiasis and septicemia.

Severe septicemia or endotoxemia may suppress the immune system by impairing cell-mediated immunity and reducing numbers and function of neutrophils.

Neonatal isoerythrolysis (NI) (Figs 11.5–11.10)

This is an immune-mediated disorder of the neonate, which results in red blood cell destruction. The foal's blood group, or alloantigens expressed on the surface of the RBC are inherited from dam and sire. If the foal has inherited an alloantigen from the sire against which the mare has produced antibodies, these antibodies, once ingested via the colostrum and absorbed into the foal's circulation, attach to the specific RBC antigen and result in intravascular or extravascular hemolysis.

During intravascular hemolysis the hemoglobin from the damaged erythrocyte is released into the blood stream, where it is bound to haptoglobulin. Once the haptoglobulin is saturated, hemoglobinemia develops. The renal glomeruli filter unbound hemoglobin, causing hemoglobinuria and the potential for renal toxicity.

In extravascular hemolysis the damaged red blood cells are removed from the circulation by mononuclear phagocytes in the spleen and liver. Hemoglobinemia and hemoglobinuria are not associated with extravascular hemolysis but icterus may be present. Extravascular hemolysis is more common in NI but intravascular hemolysis does occur.

The production of antibodies by the dam follows exposure to foreign alloantigens. This can occur due to blood or plasma transfusions, administration of products containing equine serum or exposure to fetal blood during previous pregnancies.

NI occurs most commonly in foals born to multiparous mares. These mares may have been exposed to a foreign antigen from a previous fetus due to placental hemorrhaging in late gestation or at parturition. It is thought unlikely that exposure to an antigen in late gestation will produce an antibody response in time to affect the foal from that pregnancy; it is the foals of subsequent pregnancies that are at greatest risk.

Primiparous mares may have alloantibodies, which will have been produced due to a blood/plasma transfusion or exposure to products containing equine serum.

The prevalence of NI is reported as 1% in Thoroughbreds and 2% in Standardbreds.

Clinical signs

- The foals are normal at birth with the onset of signs occurring once the colostrum has been absorbed.
- Clinical signs may become obvious at 6–12 hours of age in severe cases, but milder cases may not manifest themselves until 3–4 days old. This is due to variation in the rate and severity of the hemolysis.
- Mild signs including lethargy, tachycardia, tachypnoea and icterus will be seen in the foals which have gradual onset hemolysis and resulting mild anemia.

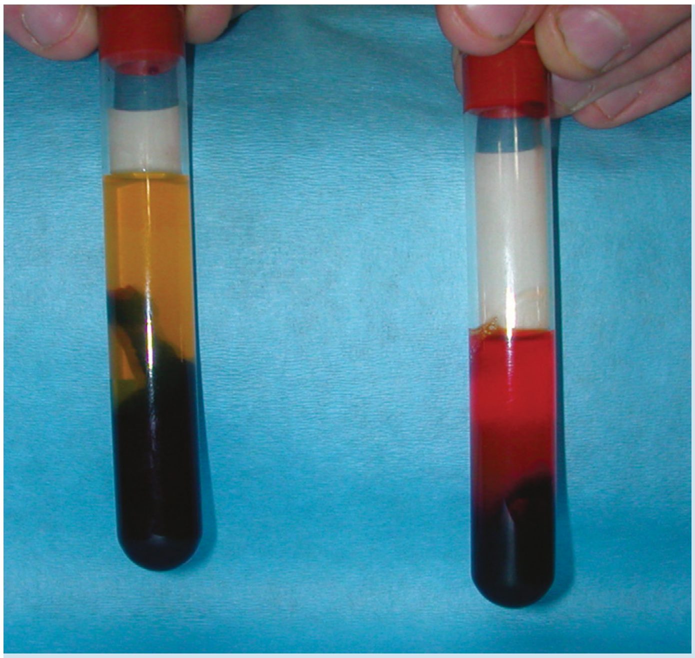


Figure 11.5: The sample on the right is a sample from a foal with NI; note the hemoglobinemia compared with the "clear" serum from a normal foal on the left. Hemoglobinemia occurs as a result of intravascular hemolysis which is much less common than extravascular hemolysis.

Figure 11.6: Pale yellow mucous membranes indicating jaundice and anemia in a foal with NI.

- The most severe signs are seen in those foals which have a rapid onset anemia due to severe hemolysis. These tend to occur earlier in the neonatal period. Signs include dullness, lethargy, tachycardia, tachypnoea, pyrexia, pale and/or icteric mucous membranes, hemoglobinuria and partial anorexia. Seizures may occur either due to hypoxia or bilirubin encephalopathy (kernicterus).

Figure 11.7: Severe jaundice as indicated by the bright yellow mucous membranes of a foal with NI.

Figure 11.8: Yellow discoloration of the mucous membranes of the vagina indicating jaundice. This is a good site for examination as subtle changes may be more obvious here than in the oral mucous membranes.

- In peracute cases of NI, there may be cardiovascular shock and hypoxia leading to multiple organ compromise and death.
- Sepsis is a common secondary problem in these foals.

Diagnosis

- The primary differentials for a neonatal foal with anemia and icterus is NI. However, icterus may not be marked on presentation in foals that have a rapid onset anemia and thus NI should be suspected in

Figure 11.9: Marked yellow discoloration of the sclera indicating jaundice in a foal with NI.

Figure 11.10: Bilirubinuria in a foal with NI.

any foal that presents with the above clinical signs and should be ruled out by laboratory tests.

- Clinicopathological findings include anemia (PCV <25%) and hyperbilirubinemia which is primarily unconjugated.
- There may be hemoglobinemia if the hemolysis is intravascular.
- Azotemia can also be present due to either dehydration, or directly as a result of the nephrotoxic effects of the hemoglobin.
- Definitive diagnosis requires demonstration of the anti-RBC antibody bound to the surface of the foal's RBC.
- The Coombs test is a direct antiglobulin test and detects antibody or complement on the surface of the RBC; however this is not specific for maternally derived anti-RBC antibodies.
- Cross-matching tests are best used prior to the foal ingesting colostrum and may aid in early diagnosis or prevention of clinical NI. These are commonly based on hemolyzing or agglutinating effects of antibody or complement binding to RBC. The presence of lysis or agglutination of its foal's RBC following ingestion of colostrum will interfere with these tests.
  - To test for hemolytic antibody, RBC from the foal and serum from the dam are mixed. Once complement is added lysis will occur if hemolytic antibodies are present in the dam's serum.
  - The jaundice foal agglutination (JFA) test, which does not require an exogenous complement source, can be carried out using basic lab equipment. It uses serial dilutions of the dam's colostrum, which are mixed with the foal's RBC, centrifuged and assessed for agglutination. This test is of greatest value when carried out prior to ingesting the dam's colostrum, to identify foals at risk of NI.

JFA test (Fig 11.11)

Materials

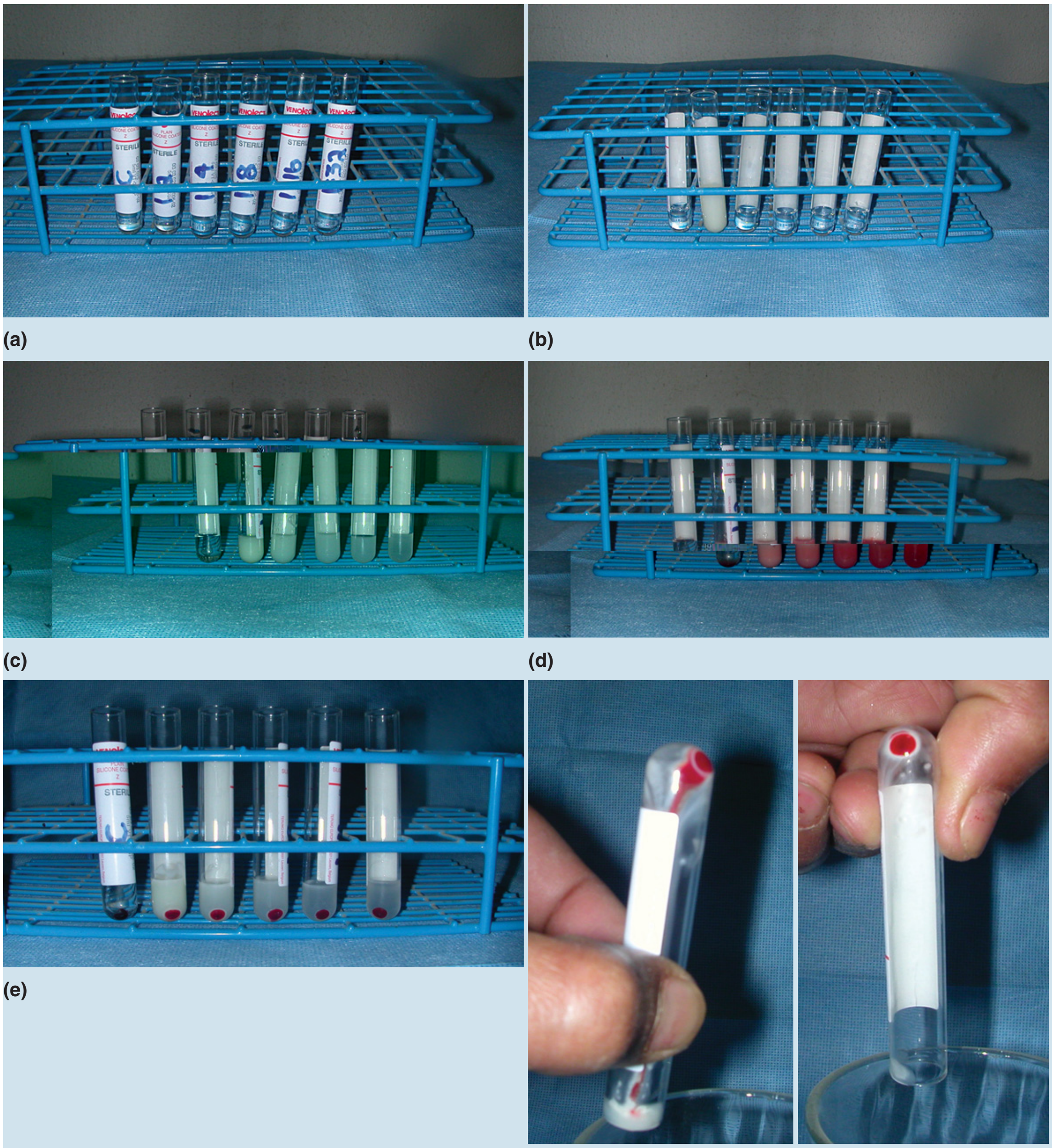
- Centrifuge
- Test tube rack
- Test tubes or red topped blood bottles
- 1 mL insulin syringes
- Isotonic (9%) saline at room temperature.

Method

- Collect colostrum or serum from the mare.
- Collect a blood sample from the foal prior to nursing in an EDTA (purple topped) tube.
- Add 1 mL of saline to 6 tubes and label as follows: Control, 1 : 2, 1 : 4, 1 : 8, 1 : 16 and 1 : 32.
- Make serial dilutions of the mare's colostrum (or serum) by adding 1 mL to the tube labeled 1 : 2 then mix, take 1 mL of the mixture and add to the tube labeled 1 : 4 and mix. Continue until all five dilutions are made and discard 1 mL from the 1 : 32 tube.
- Add one drop of the foal's whole blood to each tube and mix.
- Centrifuge the tubes for 2–3 mins at 300–600× gravity (medium speed).
- Invert each tube to pour out the liquid contents and observe the "button" of agglutination at the bottom of each tube.

Interpretation

- When no agglutination occurs, the red cells flow easily down the sides of the tube (this should happen in the control tube).
- Weak agglutination causes the cells to form small clumps as they run down the sides of the tube.
- Strong agglutination causes the cells to form large clumps.
- Complete agglutination causes the cells to remain tightly packed within the "button."
- If a positive reaction occurs with the foal's cells the test should be repeated using the mare's cells to ensure that neither the test conditions nor the viscosity of the colostrum is causing the agglutination to occur.
- Positive reactions at dilutions of 1 : 16 or greater are significant and at these dilutions correlate well with standard hemolytic assays.



(a) (b) (c) (d) (e) (f) (g)

Figure 11.11: JFA test. (a) The six tubes are labeled: C (control) and 1 : 2 to 1 : 32. 1 mL of saline is placed in each tube. (b) and (c) 1 mL of mare's colostrum is placed in the second tube (1 : 2) and then serial dilutions are made in the other tubes with 1 mL of the mixture being discarded from the final tube. (d) 1 drop of whole foal blood (EDTA) is added to each tube. (e) Following centrifugation the foal's red cells form a "button" of agglutination at the bottom of each tube. (f) and (g) The tubes are then inverted. If the red cells run freely down the side of the tube the test is negative (f). If the cells remain tightly clumped at the bottom of the tube at dilutions of 1 : 16 or greater the test is positive (g).

Differential diagnosis

The list of differentials can be varied taking into account the anemia, weakness and icterus. These include septicemia, perinatal asphyxia syndrome, perinatal herpes virus infection, hemothorax, hemoperitoneum, trauma to umbilical remnants internally or externally and drug reactions.

Treatment (Figs 11.12 & 11.13)

- Treatment of foals with NI can be variable depending on the severity of the clinical signs.
- Mildly affected foals with PCV >15% who remain bright and interested in nursing and display only mild tachypnoea or tachycardia may simply require monitoring and restriction in a stress-free and quiet environment. If identified in foals less than 24 hours of age, prevention of the foal from further nursing the dam is important. This can be achieved by using a muzzle and leaving the foal with the dam, which is less stressful than separation. An alternative source of colostrum (free of anti-RBC antibodies) and/or milk should be sourced. The mare's udder should be stripped of colostrum every 2 hours. The IgG content of the colostrum can be easily assessed at each stripping using a Brix refractometer and when the value is 10–12% it is safe for the foal to nurse. The time period when it is safe to nurse will vary considerably and can range from as low as 12 hours to greater than 48 hours depending on colostrum quality and the antibodies involved.
- The degree of anemia does not always correlate with the necessity for transfusions, as foals which gradually become anemic over the course of a couple of days often appear to compensate better cardiovascularly than foals which may have rapidly developed a less severe anemia. It is important to assess all parameters and not just the RBC indices when making the decision to transfuse.
- Foals that have a rapid decline in PCV and are showing moderate to severe clinical signs including weakness, seizures, tachycardia, tachypnoea, dehydration, azotemia, and anorexia require blood transfusions and supportive treatment.
- The ideal blood donor is one which has been blood-typed as Aa and Qa negative and has no anti-RBC antibodies. A whole blood transfusion can be given immediately without having to wait for sedimentation and separation.
- If a cross-matched donor is not available then the dam may be used as a donor. Washed RBCs from the mare are the perfect choice in that they will not react with the anti-RBC antibodies that the foal has ingested through the colostrum. After collection the whole blood must be separated and any anti-RBC antibodies removed from the RBC portion, this is done by washing the red blood cells with saline, this is usually carried out 2–3 times. This can be done at a suitable laboratory using a centrifuge to aid sedimentation. If there is no centrifuge available the blood must stand for several hours after which time the plasma can be aspirated and the red blood cells resuspended in saline. Allow to sediment, remove saline and then repeat the procedure before resuspending in *equal part* saline for administration to the foal. Without the aid of a centrifuge this can take 12–24 hours, which depending on the severity of the case may be too long. For many severe cases this is too long and if no other donor is available it may be necessary to administer the red cells resuspended in saline after a single wash.
- If an emergency transfusion is necessary and there is not a cross-matched donor available then whole blood from a healthy gelding that has no previous history of having received a blood or blood product transfusion can be used.
- 8 L of blood can be taken from a 600 kg donor without any ill-effects on the donor.
- Transfused RBCs have a short lifespan, maybe only 4–5 days, therefore repeat transfusions may be necessary in severe cases. However, one transfusion may be enough to support the foal until it starts producing new erythrocytes. Transfused cells may also sensitize the foal to future transfusions resulting in reactions. This should be considered if contemplating a second transfusion in a foal 4–7days after the first.
- 1 L of blood with a PCV of 35% would be expected to raise a 50 kg foal's PCV by 5–7%. Most foals will require 1–2 L of whole blood or resuspended red blood cells.
- Blood should be administered using specific blood administration sets, which have inline filters; if the foal is receiving concurrent intravenous medication a multilumen catheter should be used. The transfusion should be started slowly (0.5 mL/kg over 10 minutes, 25 mL in 10 min for a 50 kg foal) and the foal monitored carefully for any signs of transfusion reactions, which may include sudden elevations in respiratory or heart rates, unexplained agitation or collapse. If there is no evidence of reaction in the first period then the rate can be increased to 20–40 mL/kg/hour until the foal has received half of the required amount and then the remainder can be delivered gradually over the following 2–4 hours.
- Response to therapy can be assessed by monitoring the foals behaviour, cardiovascular status and PCV.
- A hemoglobin-based oxygen carrying solution containing polymerized bovine hemoglobin 130 g/L in solution with lactated Ringers solution (oxyglobin) has been used successfully in the therapy of NI.
  - This solution has the advantage that it is stable at room temperature, has a long shelf life and no cross-matching is required. Due to the large size of the molecules it also increases colloid oncotic pressure and therefore increases the intravascular volume.
  - Foals with NI have been reportedly treated with doses of 5–10 mL/kg with noted clinical improvement. The half life of this product in vivo in dogs in 30–40 hours. It is cost prohibitive to use it as the sole means of increasing the oxygen-carrying capacity, therefore its main use in the therapy of neonatal isoerythrolysis is to stabilize the patient while a suitable donor is being sought or the dam's red blood cells are being washed.
- Intra-nasal oxygen may help to increase hemoglobin saturation. Foals that are anorexic and weak will require assisted feeding – this is safest done via an indwelling nasogastric tube.
- Patients that are hypovolemic, dehydrated and azotemic will require polyionic fluid therapy to help restore renal function and correct electrolyte and acid–base abnormalities.
- Broad-spectrum antimicrobial therapy should be initiated prophylactically.
- Foals that are seizuring due to hypoxia or elevated levels of circulating unconjugated bilirubin will require anti-seizure therapy. Diazepam (0.1–0.2 mg/kg IV q15–30 minutes) can be used and if this is not adequate to control the seizures then phenobarbital administered as an initial loading dose of 8–16 mg/kg and followed by 5–10 mg/kg IV q8–12 h can be used. Some practitioners dilute phenobarbital in saline but this is not necessary.

Figure 11.12: Administration of a whole blood transfusion to a foal.

Figure 11.13: Oxyglobin, a hemoglobin-based oxygen-carrying solution, can be used for stabilization of NI foals while a suitable donor is found or mare's cells are being washed.

Prevention

- Identification of mares at risk of producing NI antibodies is the best means of preventing NI.
- Multiparous mares are at higher risk of producing an NI foal than a primiparous mare as they may have had foreign RBC antigen sensitization from previous pregnancies.
- Mares that have previously produced an NI foal have a 70% chance of producing another NI foal regardless whether the same sire is used or not.
- Mares should be blood typed prior to breeding or foaling – those that are Aa, Qa antigen negative are at highest risk of producing NI-causing antibodies. If these mares are bred to a stallion that is positive for Aa and Qa or the other less frequently implicated antigens, then the mare's serum should be screened for antibodies to these specific RBC antigens as close as possible to foaling i.e. within the last month.
- If the screening test indicates the presence of a weak hemolytic antibody then screening should be repeated before foaling. Once the screening test is positive for these specific antibodies then the foaling should be attended so that the foal can be muzzled to stop ingestion of the dam's colostrum.
- A JFA test or haemolytic cross match test can be carried out immediately post foaling to definitively identify if there are specific anti-RBC antibodies in the colostrum. Foals that have a positive JFA or hemolytic test should be muzzled and fed an alternative source of colostrum followed by another mare's milk, or if not available a milk substitute or goat's milk. The mare's udder should be stripped every 1–2 hours and the colostrum disposed of. The foal can be allowed to nurse when either a repeat JFA is negative or when colostrum has a Brix refractometer reading of 10–12%. If the foal is >24 hours of age and the JFA is negative at dilutions from 1 : 2–1 : 16, then it is safe for the foal to nurse.

Prognosis

- In foals that are mildly affected then the prognosis is favourable.
- Foals that have an acute onset associated with severe cardiovascular compromise and seizuring carry a poorer prognosis.
- In some foals that receive several transfusions, hyperbilirubinemia from the continuing hemolysis may cause severe kernicterus and hepatopathy; these patients have grave prognosis.
- Liver failure associated with repeated transfusions has also been reported (see Chapter 10, p. 281).

Transfusion reactions (Fig 11.14)

Two types of blood transfusion reactions may occur, anaphylactic and hemolytic:

- Anaphylactic reactions occur more commonly in animals that have received more than one blood transfusion greater than 4–7 days from the initial transfusion. The transfused erythrocytes have a 4–5-day life span in the recipient and second transfusions are safer if given within this time period, before sensitization occurs to the donor's alloantigens.
- Hemolytic reactions may occur following the transfusion if the donor's blood contains alloantibodies to the recipient's RBC antigens.

Plasma transfusions carry a low risk of reaction, especially if the plasma is harvested by plasmapheresis. However if harvested by sedimentation then it is inevitable that there will be red blood cell foreign proteins present, against which the recipient will produce alloantibodies. Home harvested plasma also carries the risk of containing endotoxin and bacteria which have the potential to cause immediate transfusion reactions.

Both blood and plasma donor horses should ideally be Aa and Qa negative, and lack any alloantibodies to RBC antigens that the recipient may have.

Major cross-matching is used to detect reactions between the donor's erythrocytes and any alloantibodies that the recipient may

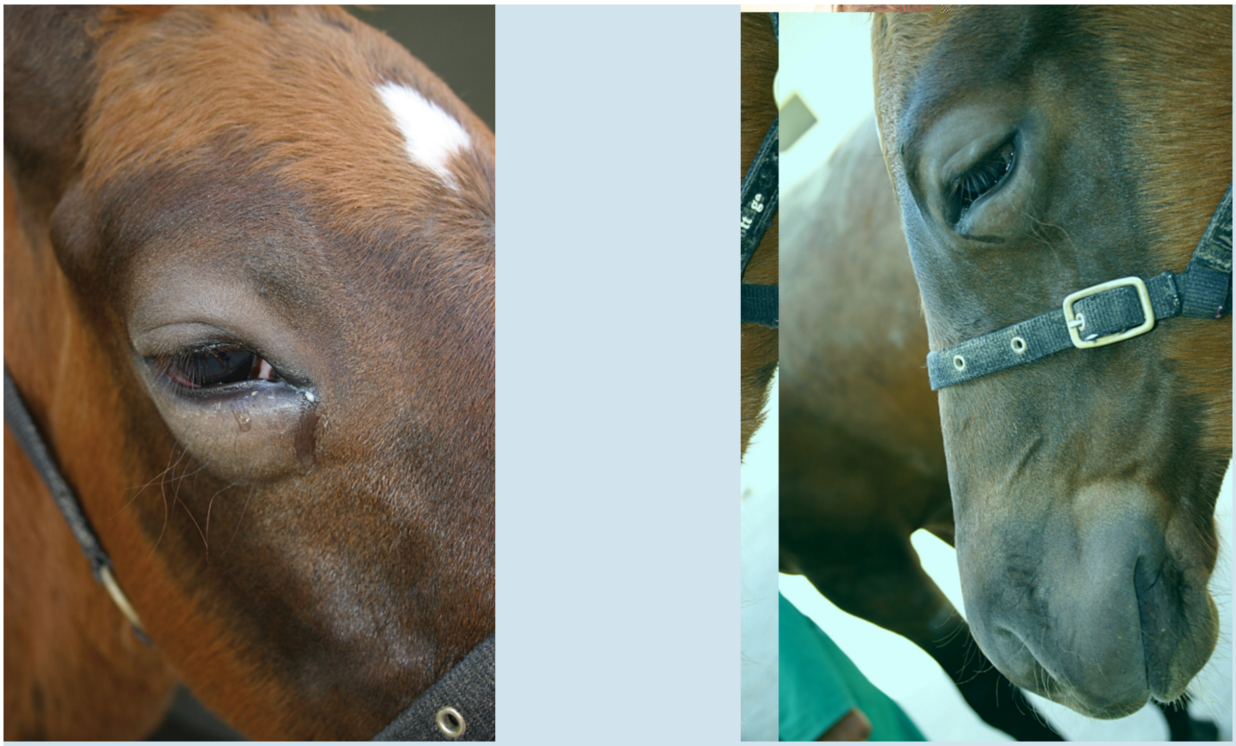


Figure 11.14: Anaphylactic transfusion reaction in a foal. Note the edema around the eyes and muzzle in this foal following a plasma transfusion. The foal had received plasma from the same batch 3 weeks previously as part of *R. equi* prophylaxis. The foal was treated with dexamethasone (0.1 mg/kg IV) and made an uneventful recovery.

have. A minor cross-match detects reactions between the recipient's erythrocytes and any alloantibody that may be present in the donor's blood. However while cross-matching will help reduce the transfusion reaction it cannot predict immune responses to other blood proteins including platelets, WBC and endotoxin if present.

If cross-matching is not available or emergency transfusion is necessary, then using a healthy gelding who has not had any previous transfusions has the least potential for reaction. A Standardbred gelding would be an excellent choice as they are negative for Qa and have a lower incidence of Aa than Thoroughbreds.

Blood and plasma should always be administered using a giving set with an inline filter and an aseptically placed catheter. Initial rates of administration should be 0.5 mL/kg over the initial 10–20 minutes while closely monitoring the patient's vital signs. If no adverse reaction is seen then this can be increased to 40 mL/kg/h with close monitoring of vital parameters. Slower rates should be used for continuous rate infusion (CRI) in critically ill foals.

Clinical signs

- Anaphylactoid-type reactions are usually immediate, and signs will include muscle fasciculations, trembling, agitation, localized or generalized edema, piloerection, tachycardia, tachypnoea and sudden collapse.
- Hemolytic crisis may present after transfusion if there are alloantibodies present in the donor's blood to the recipient's RBC cell antigens. Premature donor erythrocyte destruction may also occur if the recipient has existing alloantibodies to the donor's RBC antigens.

Treatment

- Some clinicians suggest premedicating recipients with NSAIDs or anti-histamines to reduce the incidence of reactions.
- During any transfusion it is wise to have epinephrine 1 : 1000 and dexamethasone or prednisolone sodium succinate at hand.
- If mild reactions are seen the transfusion can be temporarily ceased or the rate reduced and the patient administered dexamethasone (0.05–0.1 mg/kg IV) or prednisolone sodium succinate (1–2 mg/kg IV).
- If more severe reactions occur then stop the transfusion immediately and administer epinephrine (0.5–1.5 mL of the 1 : 1000 sol IV per 500 kg), to counteract the vasodilation and bronchial constriction that occurs in Type 1 anaphylactic type reactions. Dexamethasone or prednisolone sodium succinate should also be administered at the above doses. Colloid and crystalloid therapy may also be necessary to restore normovolemia.

Recommended reading

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CHAPTER 12

The integumentary system

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Approach to suspected conditions of the integumentary system

Skin disorders may be primarily of dermatological origin or a manifestation of disease in other organs. Diagnosis of skin disorders may be extremely challenging. The use of a dermatological examination form which allows illustration of the areas of the skin affected is useful in monitoring the progression (if any) of the disorder. History taking should include any previous illness or surgery and any medications received. A full clinical examination should be performed. The initial clinical objective should be to identify the predominant clinical sign and compile a problem list. The most common components of skin disorders are:

- pruritus
- hair loss
- scaling and crusting
- weeping and seeping
- alterations in pigmentation
- nodular lesions.

A list of differentials can then be made for each problem and used as a basis for selection of further diagnostic tests.

Diagnostic tests (Fig 12.1)

Hair sampling

Hair sampling is performed by plucking hair and associated scabs from a number of fresh lesions before and after cleaning with 70% alcohol. The samples are then placed in identified sterile containers. Hair sampling is indicated when there are broken hairs, alopecia, crusts, scales or altered growth patterns. Dermatophytosis (ringworm) and *Dermatophilus* spp infections are particularly suitable for hair sampling. Samples can then be examined directly using 10% potassium chloride (KOH) solution, cultured or in the case of Dermatophilus examined with Gram's stain.

Skin scrapings (Fig 12.2)

Skin scrapings are primarily used to identify burrowing mites (which are rare in the horse). They can also be used to identify bacterial/fungal causes of dermatitis. The area to be scraped should be lightly moistened with mineral oil.

- Superficial scrapings involve using a large blade to collect hair and macerated tissue from the superficial layers of the skin and should not cause bleeding.
- Deep scrapings involve using a large blade to collect material from the intrafollicular space and superficial dermis while holding the skin between finger and thumb. Scraping is continued until bleeding occurs.

Samples can be examined by light microscopy or can be cultured if fungal infection is a consideration.

Culture

Skin swabs for bacterial culture are unrewarding due to the large range of commensal organisms on equine skin. Culture of needle aspirates of pus from the deeper layers of the affected area may be useful. Fungal cultures of hair samples or skin scrapings can be useful.

Figure 12.1: Hair sampling – plucking hairs from the edge of a skin lesion.

Figure 12.2: Superficial skin scraping from an adult horse. Note the alopecia of the ventral elbow and inner forearm.

Sabouraud dextrose agar with a pH indicator is used and pathogenic dermatophytes cause a distinctive change in media color from amber to red in 3–5 days (before any colonies are visible).

If viral culture is to be performed the laboratory should be contacted first as special procedures for sampling and handling may be required.

Biopsy (Figs 12.3–12.5)

Biopsy specimens can be used for:

- histological diagnosis
- needle aspirate cytology
- special tests such as virus identification, bacterial/fungal swabs, or immunohistochemistry/immunofluorescence.

The type of biopsy (shave, punch or wedge) will depend on the disorder present and the pathologist should be consulted as early as possible. It is important to remember that the response of the skin to a variety of insults can be histopathologically similar and thus results may be inconclusive.

It is important not to sterilely prepare the area before sampling. Certain disorders such as pemphigus foliaceous may not be detected if the area is sterilely prepared. Once the biopsy has been taken then the site should be sterilely prepared before the placement of sutures or staples.

Figure 12.3: A shave biopsy being taken from the bulb of a heel of a horse with suspected pemphigus foliaceus. The area has been anesthetized with a palmer digital nerve block (from Pascoe RR, Knottenbelt DC 1999 Manual of equine dermatology. Saunders, London).

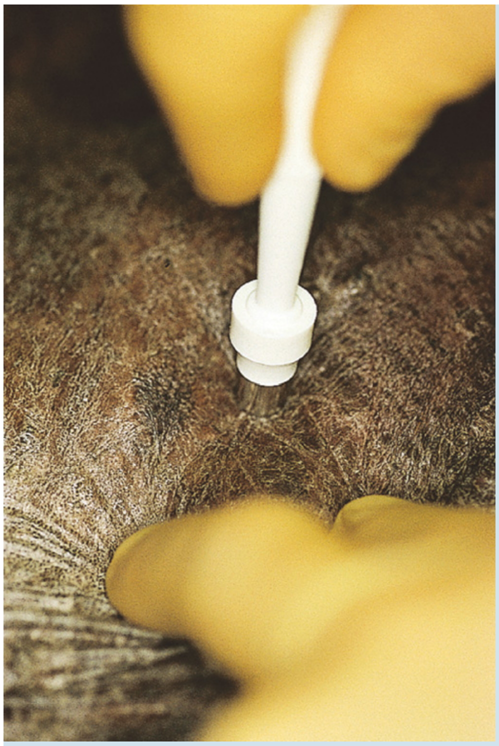


Figure 12.4: A skin biopsy sample being taken with a disposable biopsy punch. Systemic sedation and analgesia is generally all that is required and it is not necessary to suture the site (from Pascoe RR, Knottenbelt DC 1999 Manual of equine dermatology. Saunders, London).

Figure 12.5: A wedge biopsy being taken. The site was closed with skin staples (from Pascoe RR, Knottenbelt DC 1999 Manual of equine dermatology. Saunders, London).

Figure 12.6: Epitheliogenesis imperfecta in a 1-day-old foal.

Inherited / congenital conditions

Epitheliogenesis imperfecta (Fig 12.6)

This is a rare inherited and congenital defect which has been documented in Belgium Draft horses and American Saddlebred horses.

Figure 12.7: Epidermolysis bullosa in a Belgian foal (from Pascoe RR, Knottenbelt DC 1999 Manual of equine dermatology. Saunders, London).

Clinical signs

- Affected foals have clearly demarcated areas with a complete absence of epidermis present at birth.
- Lesions bleed easily and quickly become infected, leading to septicemia and death.

Diagnosis and differentials

- Clinical appearance and a negative Nikolsky's sign (transverse finger pressure does not cause the epidermis to "slip" off the underlying dermis).
- Epidermolysis bullosa is the only differential for this condition and will produce a positive Nikolsky's sign.

Treatment

- Small lesions may be closed with repeated surgical intervention. However, most cases cover extensive areas making treatment impossible.
- The owner should be counseled about the heritable nature of the disorder.

Epidermolysis bullosa (Fig 12.7)

Epidermolysis bullosa is seen predominantly in Belgian foals and is characterized by blister formation following mild trauma.

Clinical signs

- Lesions are commonly seen on the oral mucosa, at mucocutaneous junctions and elsewhere, and are present at birth.
- Additional lesions develop shortly after birth.

- Separation of the hooves at the coronary bands is sometimes present.

Differential diagnosis

- Epitheliogenesis imperfecta
- Pemphigus foliaceus
- Scalding and burn injuries.

Diagnosis

- History and clinical appearance are very suggestive of the condition.
- Histopathology of skin biopsies demonstrates cleavage at the dermal–epidermal junction.
- Positive Nikolsky's sign (transverse finger pressure causes the epidermis to "slip" off the dermis).
- A polymerase chain reaction (PCR) test has been developed to identify carriers of the mutation using mane and tail hairs. Since October 2002, this test has been available to breeders of Belgian horses in the United States and Canada through their breed associations.

Treatment

There is no treatment and the owners should be counseled about the heritable nature of the disorder.

Dentigerous cyst (Fig 12.8)

See Figures 4.10 & 4.11 p. 84.

Figure 12.8: Dentigerous cyst with draining tract in a typical location at the base of the ear.

Dermoid cyst (Fig 12.9)

Dermoid cysts are hereditary cutaneous cysts most commonly seen on the dorsal midline between the withers and rump.

Clinical signs

- Single or multiple, smooth dermal nodules with normal overlying hair.
- Lesions contain a soft cheese like material and sometimes coiled hairs.
- Usually seen in horses aged 1–3 years but can be seen earlier.

Differential diagnosis

- Epidermoid cyst
- Dentigerous cyst
- Hypodermiasis.

Diagnosis

- Location and appearance is highly suggestive
- Excisional biopsy.

Treatment

Surgical ablation of cyst wall and contents is effective.

Epidermoid cysts (atheroma) (Fig 12.10)

These are most commonly seen just above the caudal limit of the false nostril and in this site are known as nasal atheroma. Other sites that can be affected are the limbs and head. They may be evident at birth or more commonly become apparent after a number of months.

Figure 12.9: Two dermoid cysts on the dorsal midline of a 13-month-old colt, these had been evident since the colt was 3 months of age.

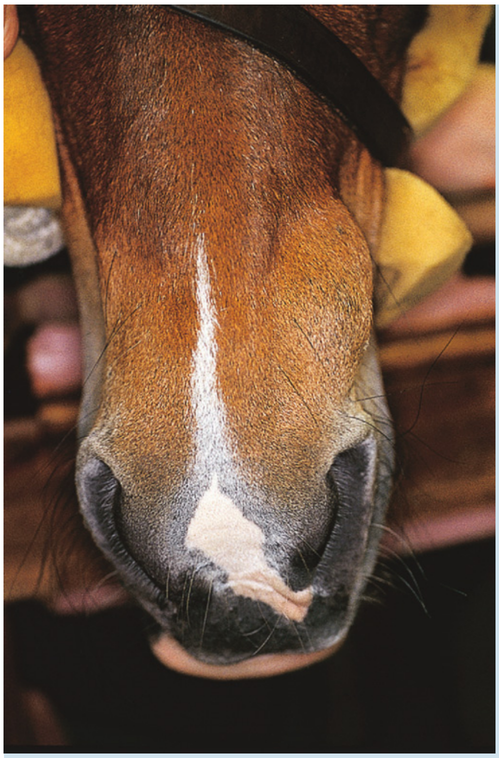


Figure 12.10: Atheroma (nasal inclusion cyst) in a typical location (from Pascoe RR, Knottenbelt DC 1999 Manual of equine dermatology. Saunders, London).

Clinical signs

- Well circumscribed, freely movable solitary nodules.
- They contain a yellow to grey mucoid fluid with no hairs and usually remain static for years.
- Rarely the cyst may reach a size sufficient to interfere with airflow in the affected nostril.

Differential diagnosis

- Dentigerous cyst
- Dermoid cyst
- Foreign body reaction
- Nodular sarcoid.

Diagnosis

- Clinical signs and aspirate appearance are highly suggestive.
- Histopathology of biopsy specimens shows an epithelial lining but no adnexal structures.

Treatment

Surgical removal is effective.

Figure 12.11: Loose, hyperextensible skin over the hip area, typical of HERDA.

Figure 12.12: Easily torn skin with non-healing wounds in a HERDA colt.

Hereditary equine regional dermal asthenia (HERDA) (Figs 12.11–12.13)

History

This condition is also known as cutaneous asthenia, hyperelastosis cutis and Ehlers-Danlos syndrome. It is seen primarily in Quarter-horses but has been recognized in other breeds. Affected horses generally develop "loose skin" before 6 months of age but in many cases it may not be recognized until later in life. Pedigree analysis has demonstrated an autosomal recessive mode of inheritance.

Clinical signs

- Loose, wrinkled or hyperextensible skin over three or more areas of the body (usually dorsolaterally).
- The skin tears easily and is slow to heal.

Figure 12.13: Chronic non-healing wound on a HERDA filly.

Diagnosis

- The combination of a suitable history and clinical signs is suggestive of the condition.
- Biopsies are currently required to confirm the diagnosis. Incisional full-thickness biopsies should be taken, as punch biopsies may not contain deep dermis.
- A diagnostic DNA test is available through the University of California at Davis. Hair samples from the mane or tail with intact roots are required.

Treatment

There is no treatment other than supportive care. The owner should be advised of the heritable nature of the condition.

Cutaneous hemangioma / vascular hamartoma (Fig 12.14)

These are congenital cutaneous tumors of blood vessels which have been most frequently reported in Arabians. Commonly affected sites include the groin, thorax, distal limbs and elbow.

Clinical signs

- The congenital form has a fleshy, proliferative appearance and is restricted to the skin surface. Minor trauma causes bleeding.
- Larger vascular hamartomas and hemangiomas may involve the overlying skin and can be found in subcutaneous tissues.

Treatment

Surgical removal followed by skin grafting can be attempted but there is a high failure rate. Frequently the location of the lesion makes complete surgical removal difficult.

Figure 12.14: Cutaneous hemangioma in the groin area.

Figure 12.15: Congenital papilloma on the forehead of a newborn foal.

Congenital papillomas (Figs 12.15–12.17)

The papilloma virus has the ability to cross the placenta and foals can be born with single cauliflower-like warts on the head, neck or trunk.

- Diagnosis is made on clinical appearance as no other condition appears similar.
- Ligation and surgical removal is generally effective although some resolve spontaneously.

Acquired conditions

Anagen defluxion / Telogen defluxion (Fig 12.18)

Shedding of the foal coat normally occurs at 3–4 months of age. During this process there may be areas of decreased coat density and

Figure 12.16: Large congenital papilloma on the lower lip of a newborn foal.

Figure 12.18: Anagen defluxion characterized by regional hair loss.

Figure 12.17: Large congenital papilloma after removal from a newborn.

occasionally alopecia, both of which are normal. Anagen defluxion occurs when a special circumstance (fevers, illness etc.) interferes with the anagen phase of the hair cycle, resulting in abnormalities of the hair follicle and shaft. Hair loss occurs *within days* of the insult.

Telogen defluxion occurs when a stressful situation (surgery, pregnancy, fevers, shock etc.) causes the abrupt, premature cessation of hair growth of many anagen hair follicles and the synchronization of these hair follicles in catagen and then telogen. Hair loss begins within *1–3 months of insult.*

Diagnosis

- The age of the foal allows differentiation from other congenital causes of alopecia such as epitheliogenesis imperfecta and epidermolysis bullosa.
- History can also rule out causes of alopecia such as contact dermatitis.
- Skin biopsy is rarely helpful.
- Direct hair examination (trichogram) with a light microscope allows assessment of the growth pattern of hair (telogen vs anagen).

Autoimmune conditions

Pemphigus foliaceus (Figs 12.19–12.21)

Pemphigus foliaceus is an autoimmune skin disease characterized by an exfoliative dermatitis. It has been seen in a variety of breeds. Signs can be seen from 2 months of age.

Clinical signs

Early cases

- There are transient vesicles and pustules with superficial erosions. These usually go unnoticed.
- Scaling and crusting commonly begins on the face or limbs and extends to the rest of the body over a period of days to weeks.
- Extension of the lesions leads to marked alopecia but there is usually little if any pruritus in the early stages.
- In cases in which pruritus occurs, secondary lesions may be misleading and can also interfere with biopsy interpretation.

Advanced/chronic cases

- Scaling and crusting is extensive and severe resulting in marked alopecia.
- Systemic signs will also be seen and include weight loss, fever, limb edema and lethargy.

Differentials

- Dermatophilosis
- Dermatophytosis


Figures 12.19 & 12.20: Scaling and crusting on the face of a foal with pemphigus foliaceus.

Figure 12.21: Same foal as in Figs 12.19 & 12.20, showing extension of the crusted lesions over the body.

- Staphylococcal folliculitis
- Drug eruptions
- Chemical dermatoses
- Seborrhea.

Diagnosis

- History and clinical signs are highly suggestive
- Biopsy
- Immunofluorescence testing.

Treatment

- Juvenile onset (<1 year) cases generally respond better to therapy and are more likely to remain in remission after treatment is withdrawn.
- The initial treatment of choice is either prednisolone (1–2 mg/kg PO q24h or dexamethasone (0.05–0.1 mg/kg PO q24h).
- The induction dose is maintained until the disease is inactive (normally 10–14 days).
- The dose is then tapered to an alternate morning regimen over a further 10–14 days. Total treatment times in affected foals are commonly 6–8 weeks.
- Patients that fail to respond to the above may respond to higher doses of dexamethasone. Doses of as high as 0.4 mg/kg PO q24h have been used in some poorly responsive cases. The lowest effective dose should be used.
- If neither of the above is effective, treatment with gold salts may be attempted.
- Other important aspects of treatment are sun avoidance and gentle skin cleansing in pruritic cases.

Other autoimmune skin disorders have been reported in animals less than one year of age but are rare. The reader is referred to the list of recommended reading at the end of this chapter for information on other such disorders.

Figure 12.22: Severe necrotizing dermatitis in a foal with diarrhea.

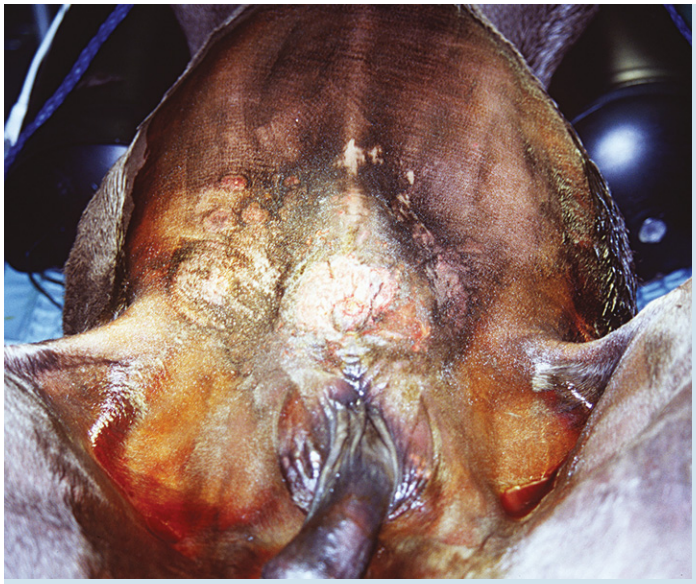


Figure 12.23: Contact dermatitis as a result of the use of a strong iodine concentration.

Dermatitis (Figs 12.22–12.24)

Inflammation of the skin (dermatitis) is most commonly caused by initial skin wetting and maceration and can be followed by infection. It is most commonly seen in recumbent foals that are in contact with urine or body exudates. It is also frequently seen in foals with diarrhea. Contact dermatitis can also occur as a result of the use of chemical agents, e.g. strong concentrations or overuse of iodine. *Streptococcus* and *Staphylococcus* spp are most frequently isolated but *Dermatophilus congolensis* can also be seen in older foals in wet conditions.

Clinical signs

Signs can vary from mild exfoliation to extensive areas of alopecia with exudation and crusting.

Diagnosis

- History and clinical signs are usually sufficient for a diagnosis.
- Bacterial culture may be useful to identify the organism involved.
- Skin scrapings and/or biopsies may be useful in some cases.

Treatment

- Treatment depends on the severity and duration of the dermatitis.
- In cases of contact dermatitis the initiating cause should be identified and eliminated.
- Mild cases that are not yet infected can be treated by maintaining a dry surface. This may include clipping and frequent changes of bedding in recumbent foals. Foals with diarrhea should have their perineum and hind quarters cleaned and thoroughly dried on a regular basis. Antibacterial and petroleum-based creams may help to keep the skin dry and prevent further damage.

Figure 12.24: Severe Staphylococcus dermatitis of the hindlimbs in a septic foal.

- Gentle antibacterial washes may be helpful in mild cases but care should be taken to avoid strong chemicals which may further induce a contact dermatitis.
- Severe cases may require systemic antibiotic therapy.

Decubitus ulcers / pressure necrosis (Figs 12.25–12.27)

- Pressure necrosis results from prolonged recumbency or destruction of the skin blood supply from applied pressure (bandages, casts).

Figure 12.25: Pressure sore over the lateral hock in a HIE foal.

Figure 12.26: Early pressure wound over the lateral hip, note the subtle change in hair color when compared with the surrounding area. At this point the area would be palpable firmer than the surrounding tissue.

- Some pressure wounds can be very slow to appear. There may be an area of pain, heat or swelling for a number of days or weeks before the necrosis becomes apparent.
- Treatment of such wounds whether on the body (associated with recumbency) or on the distal limbs (associated with bandaging) is difficult and prolonged.
- Healing by granulation and epithelialization is slow and may result in extensive scarring.
- Secondary complications such as infection and septicemia or damage to tendons can be serious and even life threatening.

Figure 12.27: Advanced pressure sore over the hip area.

Figure 12.28: Extensive folliculitis seen as tufted papules.

Folliculitis and furunculosis (Fig 12.28)

Folliculitis is inflammation (with or without infection) of the hair follicles. When the inflammatory process includes the hair follicle and surrounding tissue it is known as furunculosis. The most common causes are infections with *Staphylococcus* spp and *Corynebacterium pseudotuberculosis*.

Clinical signs

- Folliculitis most commonly appears as tufted papules. These may then progress to develop a central ulcerated area that discharges a serosanguinous fluid before becoming encrusted in the healing phase.

- Furunculosis is characterized by a combination of nodules, draining tracts, ulcers and crusts. Localized edema and cellulitis may even occur.
- Lesions are most common in the spring and autumn.

Diagnosis

- History and clinical signs are usually sufficient for diagnosis.
- Culture of deep tissues may be performed, especially if *Corynebacterium sp.* is suspected.
- Biopsies are not commonly performed.

Treatment

Treatment varies with the severity and duration of infection and can include:

- hair clipping to remove matted hair and debris, improve drainage and allow better penetration of topically administered agents
- topical applications of chlorhexidine or benzoyl peroxide
- combined topical and systemic antibiotics for more extensive or severe lesions – the length of treatment may be prolonged as antibiotics are generally continued for at least one week following visual and palpable healing of deep lesions.

Dermatophytosis / ringworm (Figs 12.29–12.31)

History and clinical signs

- Ringworm is a highly contagious fungal infection that affects horses of all ages.
- Younger horses, especially foals, are less resistant to infection and also take to longer to recover.
- The infective spores are highly resistant to environmental conditions and can survive on tack, on fence posts and in stables for years.
- Most cases of ringworm are caused by *Trichophyton* spp. The earliest sign, which is commonly missed, is a raised circular area of hair from which the hair can be easily plucked. These areas then merge to produce large irregular areas of alopecia with marked scaling. Occasionally exudates and crusting may also be seen.
- *Microsporum* spp infections are less common and usually less severe.

Diagnosis

- History and clinical signs are highly suggestive especially in an outbreak situation.
- Cultures can also be used to obtain a diagnosis.

Treatment and control

- Treatment is the same regardless of the organism involved so differentiation is usually not required.
- The disease usually resolves spontaneously in 6–12 weeks and exposure to direct sunlight may shorten the course.
- Treatment is generally undertaken to limit the spread of infection and includes use of antifungal agents such as enilconazole, natamycin and miconazole depending on local regulations and availability. These products are normally applied as washes over the entire horse.
- Systemic administration of fluconazole or itraconazole may be of benefit in areas where topical washes are unavailable.
- The use of oral griseofulvin has not been proven to be efficacious but there are anecdotal reports of its benefit.
- Prevention of spread can be difficult. Methods of environmental treatment include careful disposal of all infected hairs and scabs

Figure 12.29: Circular areas of alopecia with scaling in a foal with dermatophytosis.

Figure 12.30: Same foal as in Fig 12.29 showing large coalescent areas of alopecia in addition to well demarcated circular areas of alopecia.

Figure 12.31: Dermatophytosis on the face of an Arabian foal. Note the large area of alopecia with marked crusting near the muzzle and the alopecia with crusting around the eye.

Figure 12.32: Myiasis of a wound. Note the many larvae in and around the wound.

Figure 12.33: Myiasis of a recently opened caslick prior to foaling. This was treated with topical and oral ivermectin in addition to flushing and healed well.

by burning, stable fogging with enilconazole or potassium monopersulphate and fumigation of tack with formaldehyde.

Myiasis (Figs 12.32 & 12.33)

Myiasis is infestation of tissue by fly larvae. Blow-fly strike (*Calliphora* spp) is most common and usually affects discharging, infected or neglected wounds and can be seen in any age foal.

Clinical signs

- The affected wound is malodorous.
- The presence of larvae within the tissue is extremely irritating and the animal will often chew and lick the affected area.

Diagnosis

Close examination of the wound will reveal the presence of larvae, which are often located deep within the wounds.

Differentials

- Exuberant granulation tissue.
- Fibroblastic sarcoid.
- These conditions can also be secondarily affected by fly strike which can result in significant pruritus in conditions that are not normally pruritic.

Treatment

- Clean and irrigate the wound thoroughly.
- Remove all visible larvae.
- In very deep wounds, it may not be possible to remove all the larvae at once and repeated flushes at 12–24-hour intervals may be required.
- Ivermectin can be applied topically. The paste form is easy to apply.

Pediculosis / louse infestation (Figs 12.34 & 12.35)

Lice are host-specific, obligate parasites. There are two major groups:

- biting lice (*Damalina equi*) feed on surface scales and are deep brown in color
- sucking lice (*Haematopinus asini*) feed on blood, are larger and are yellow brown in color.

Spread is by direct contact or indirectly from the stable environment or contaminated equipment such as grooming brushes. Disease is

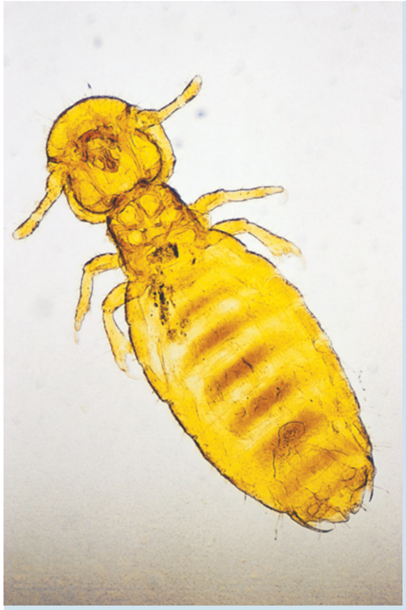


Figure 12.34: *Damalina equi* louse (from Pascoe RR, Knottenbelt DC 1999 Manual of equine dermatology. Saunders, London).

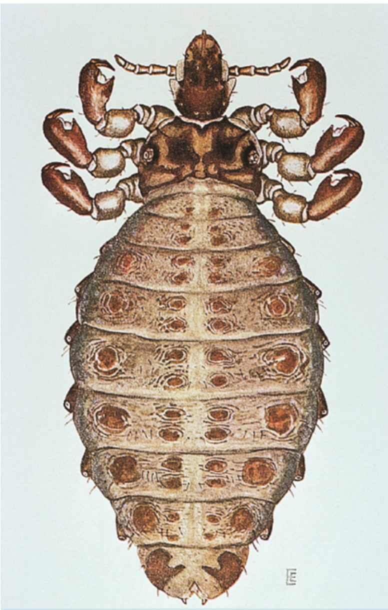


Figure 12.35: *Haematopinus asini* louse (from Pascoe RR, Knottenbelt DC 1999 Manual of equine dermatology. Saunders, London).

most common in the winter months with dam and foal affected concurrently.

Clinical signs

- Biting lice prefer the back and sides of the neck.
- Sucking lice are found mainly on the neck, tail and limbs.
- Lice of all types cause pruritus which results in rubbing, dermatitis, secondary alopecia.
- Loss of condition can be marked in severely affected cases.
- Sucking lice can cause a clinically significant anemia.

Diagnosis

- Sucking lice can often be readily identified with the naked eye.
- Biting lice are smaller and their eggs are laid closer to the skin, making them harder to identify.
- Groomings can be examined on a dark tile where the parasites can be seen to move.
- Examination with a light microscope will identify the species. *Damalina* spp have a relatively broad body with a squarish head while *Haematopinus* spp have a longer, narrower body and a conical head.

Differentials

- Dermatophilosis.
- Chorioptic and psoroptic mange (may be seen in older weanlings).
- Trombiculosis/harvest mite infestation. Disease is most common in late summer and fall when larvae are active. Larval attachment is most common on the muzzle, face, ears, neck and distal limbs as the horse grazes. Lesions consist of papules and wheals and close examination of early lesions reveals a red/orange larva at the center of the lesion. It can be differentiated from pediculosis by the time of the year and occurrence in horses at pasture.

Treatment

- Pyrethroid washes such as cypermethrin and permethrin can be useful and give more consistent results than pyrethroid powders or spot-on treatments.
- Oral ivermectin has a limited effect but pour-on preparations may be more useful. The manufacturer's recommendations relating to dosage, age at which they can be administered, repeat doses and concurrent drug therapy should be followed closely.
- Affected and in contact horses should be treated at 10-day intervals.
- Successful treatment results in cessation of pruritus within 24–36 hours.

Sarcoids (Fig 12.36)

Sarcoids are rare in foals but they can occur. The verrucous and fibroblastic forms are the types most frequently identified. There is usually a history of a chronic non-healing wound and the mare or other in-contact horses may have sarcoids.

Diagnosis

Diagnosis can be difficult and biopsies which are characteristic carry the risk of exacerbation and thus should only be performed when there is a definite treatment plan should the biopsy confirm the diagnosis.

Treatment

- Treatment is difficult as surgical excision is not possible in a lot of common sites such as the distal limb.

Figure 12.36: Fibroblastic sarcoid in the pastern region of a 3-week-old foal (from Knottenbelt DC, Holdstock N, Madigan J 2005 Equine neonatology medicine and surgery. Saunders, London).

- Topical cytotoxic drugs are not suitable for use in young foals.
- Some cases have been reported to self-heal.

Papillomatosis / grass warts (Figs 12.37 & 12.38)

- Grass warts are usually seen as multiple single or coalescing pink or grey vegetative lesions on the muzzle, lips, face, distal limbs and genitalia. They occur most commonly in grazing animals from 6 months to 4 years.
- They are moderately contagious by direct and indirect contact.
- Diagnosis is generally made on the basis of clinical appearance but biopsy can be performed.
- Treatment is not usually required as most lesions on young horses resolve spontaneously in 3–4 months. Autogenous vaccines can be used but due to the time delay in preparation there may be no clinical advantage. Topical podophyllium (used for treatment of plantar warts in humans) can be used for more rapid resolution.

Figures 12.37 & 12.38: Viral papillomas on the muzzle and around the eye of a yearling filly.

Recommended reading

Pascoe RR, Knottenbelt DC 1999 Manual of equine dermatology. Saunders, London

Scott DW, Miller WH 2003 Equine dermatology. Saunders, London

CHAPTER 13

The endocrine and metabolic systems

Siobhan B. McAuliffe MVB DACVIM

Normal function of the endocrine system is important not only in intrauterine development and adaptive processes following birth but also in the normal function of other body systems. Endocrine and metabolic disorders are frequently overlooked as physical evidence of malfunction may not be apparent.

Endocrine disorders

Neonatal hypoadrenocorticism

History and clinical signs

The appropriate physiologic response to stressors such as severe systemic illness involves activation of the hypothalamic–pituitary–adrenal (HPA) axis, ultimately resulting in release of cortisol into the systemic circulation in amounts appropriate for the degree of physiologic stress. Cortisol is one of the body's main "stress hormones", and has many beneficial systemic effects that enable the body to respond to the stresses of disease. However, in many critically ill humans, the adrenal gland cannot appropriately respond to the stress of severe illness, a diagnosis termed "relative adrenal insufficiency" (RAI). Patients with RAI have inappropriately low serum cortisol levels for their degree of illness and display abnormal responses in adrenal function testing.

Normally, the HPA axis tightly regulates systemic cortisol levels in both health and disease, and thus plays an integral role in the maintenance of cellular, organ, and whole body homeostasis. Both external and internal environmental stressors activate the peripheral and central nervous systems, whose signals are interpreted and integrated in the hypothalamus, culminating in the release of corticotropin-releasing hormone (CRH). CRH acts locally on the anterior pituitary gland, inducing the release of adrenocorticotropic hormone (ACTH or corticotropin). ACTH, in turn, enters the systemic circulation and stimulates the adrenal cortices to release cortisol. Increased systemic cortisol levels then exert a negative feedback effect on both the pituitary gland and hypothalamus, resulting in subsequent down-regulation of both CRH and ACTH secretion. With an intact HPA axis, systemic cortisol levels are maintained at a level that is appropriate for the existing degree of physiologic stress.

Much less is known about adrenal function and dysfunction in equine neonates. However, HPA axis function and cortisol response in the perinatal period has been evaluated in healthy foals. Compelling evidence shows that maturation of the HPA axis starts during the last 4–5 days prior to parturition and continues into the first 1–2 weeks of life. For example, premature foals (gestational age <320 days) have low levels of serum cortisol (<3 μg/dL) in the 2 hours after birth, as compared to term foals (12–14 μg/dL). Concurrently, premature foals showed significant elevations in endogenous ACTH levels, with peaks of 650 pg/mL at 30 minutes postpartum, as compared to a peak of 300 pg/mL in term foals. This low baseline cortisol and delayed, high ACTH peak in premature foals suggest that the problem with cortisol secretion resides at the level of the adrenal gland. Corticotropin stimulation tests (125 μg cosyntropin IM) support this theory, as premature foals showed a poor response to ACTH, with only a 28% increase in plasma cortisol 30–60 minutes following stimulation, as compared with a 208% increase in normal term foals. In addition, premature foals in this study showed other abnormalities consistent with adrenal insufficiency, including abnormally low neutrophil to lymphocyte ratios, hypoglycemia and hypoinsulinemia.

Diagnosis

- Neutrophil to lymphocyte ratio of 0.5–1.0 without bands or immature neutrophils.

- Lack of response to ACTH challenge.
- High serum ACTH levels and low serum cortisol levels indicate adrenal insufficiency.

Differential diagnosis

- Prematurity/dysmaturity with normal adrenal function
- Hypoxic–ischemic encephalopathy
- Hypoglycemia
- Septicemia.

Treatment and prognosis

- Normal function may occur with time and supportive care.
- Intramuscular injection of depot tetracosactrin (ACTH) has been used to accelerate adrenal maturation. Replacement of cortisol may be indicated until normal adrenal function has been stimulated by depot injections.
- Adrenal function will usually attain normal levels; however the degree of prematurity/dysmaturity and severity of associated problems (hypoplasia of carpal and tarsal bones) will often determine the overall outcome of such cases.

Hypothyroidism (Fig 13.1)

Etiology and history

Hypothyroidism in foals may be associated with goiter (abnormal enlargement of the thyroid gland) or may occur in foals with grossly normal thyroid glands. The most common cause of goiter in neonatal foals is hyperiodic hypothyroidism and is most commonly related to feeding of iodine rich supplements (often seaweed-based products) to the mare during pregnancy. The mare may show no signs and can in fact be euthyroid.

Thyroid hormones are required for mental activity/neural development, lung maturation, gastrointestinal function, cardiovascular function and the growth/maturation of the skeletal system.

Figure 13.1: Hypothyroidism in a foal with marked enlargement of the thyroid gland.

Hypothyroidism in foals has been separated into two categories by some authors:

1. Hypometabolic state which is seen concurrent with low thyroid hormone levels.
2. Developmental lesions, which can be seen in conjunction with normal thyroid hormone levels and are thus an indication that thyroid hormone inadequacy occurred at a critical point in development.

Clinical signs

- Foals may be born with no apparent clinical signs, but skeletal lesions generally appear within a few weeks and include marked angular limb deformity of the carpus and/or "sickle-hocked" appearance with "bunny-hopping" gait secondary to incomplete ossification of the tarsus.
- Goiter; both thyroid glands may not be uniformly enlarged.
- Other clinical signs are less specific and it may often be difficult to differentiate from congenital lesions, without a specific history. These signs include:
  - weakness, dysmaturity or low birth weight
  - incoordination, poor suckling and righting reflexes
  - hypothermia
  - skeletal abnormalities including poor bone ossification, tendon contracture or laxity, rupture of the common digital extensor tendon and mandibular prognathia
  - thin skin and poor hair coat
  - organ system immaturity.

Diagnosis

- Presence of goiter
- Thyroid hormone estimations:
  - T_3 and T_4 levels are high in normal foals at birth and decrease over the first 4 weeks of life to adult levels. Estimation of thyroid hormone levels from foals suspected of hypothyroidism should be made in conjunction with an age-matched control.
  - TSH or TRH stimulation tests are not often performed due to the expense and difficulty in obtaining exogenous forms of the hormones.
  - Beware of over interpretation as *thyroid hormone levels can be normal, even in foals with goiter*.
- Biopsy of the thyroid gland to demonstrate hyperplastic changes.

Treatment

Obtaining a definitive diagnosis may be an academic point as supplemental thyroid hormones have not proved helpful and as such there is no treatment other than supportive care.

Prognosis

The prognosis is poor for all except the mildest cases. Many affected foals die at an early age. Of those with skeletal abnormalities, only a few will reach the desired athletic performance.

Idiopathic hypocalcemia

History and clinical signs

Reported cases have ranged in age from 4 days to 5 weeks and presented with tachycardia, hyperhidrosis, seizures, recumbency, diarrhea or a recent history of diarrhea and muscle rigidity or a stiff gait.

Diagnosis

Total serum calcium concentration <6.5 mg/dL.

Differential diagnosis

- Acute or chronic renal failure (hypercalcemia is more common). Can be differentiated by normal serum creatinine and blood urea nitrogen (BUN) concentrations.
- Parathormone (PTH) resistance (has not been shown to occur in foals but is a familial disorder in humans).
- Hypocalcemia associated with septicemia (differentiated by normal IgG levels and absence of other associated signs of septicemia).
- Insufficient absorption of calcium by the gastrointestinal tract. This may occur during or following diarrhea but is not associated with chronic hypocalcemia.
- Inadequate dietary intake of calcium, excess dietary phosphorus or magnesium deficiency (unlikely in nursing foals).
- Primary hypoparathyroidism.
- Hypoalbuminemia secondary to protein-losing enteropathy

Treatment and prognosis

- Foals with idiopathic hypocalcemia were treated with intravenous calcium but failed to respond to treatment and either died or were euthanized.
- Transient hypocalcemia associated with septicemia or decreased intestinal absorption following diarrhea generally responds well to supplemental calcium.

Hyperparathyroidism (nutritional secondary hyperparathyroidism) (Figs 13.2–13.5)

History

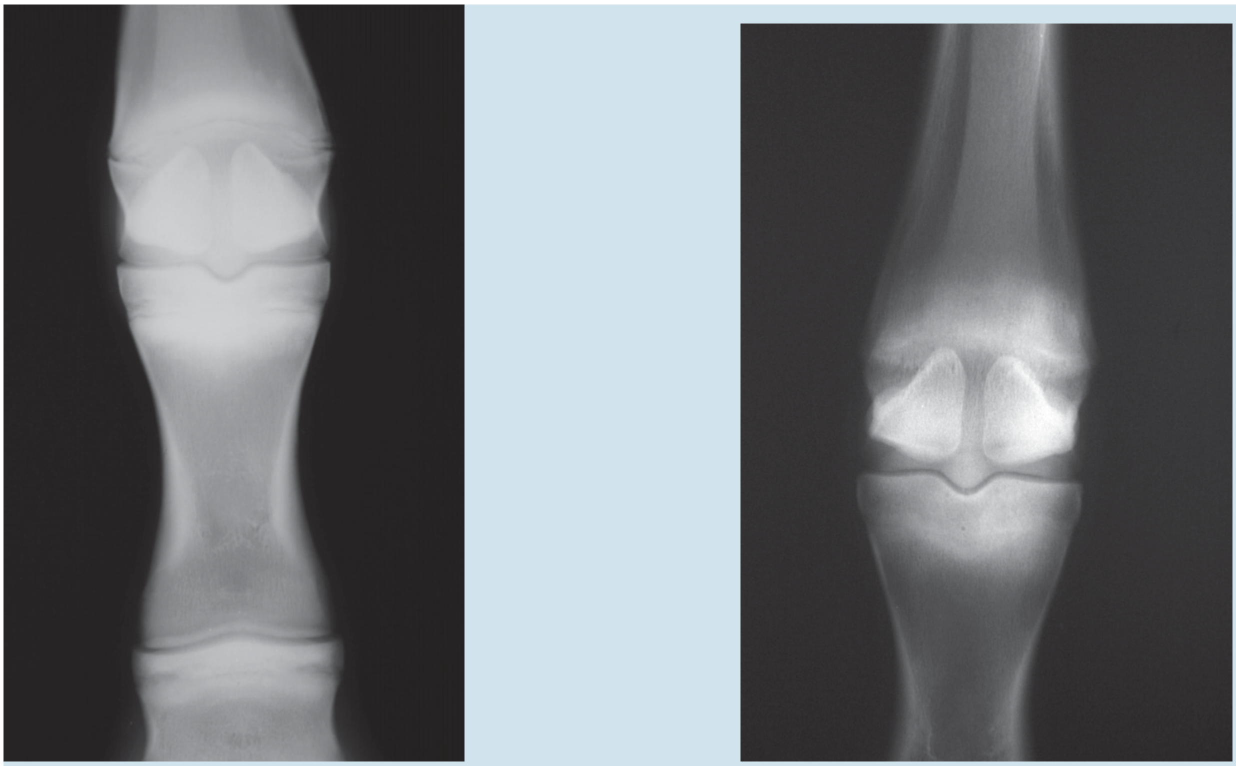
This condition is now rarely reported in developed countries but is perhaps underdiagnosed in other regions. In the author's experience the condition is common in desert countries. It is associated with diets low in calcium or high in phosphorus, or both, and is most commonly associated with grain-rich diets. It can also be related to high dietary intake of oxalates which bind calcium.

Clinical signs

- Clinical signs may be subtle and easily confused with other conditions.

Figures 13.2 & 13.3: Foal with nutritional secondary hyperparathyroidism. This 8-month-old foal presented 2 weeks prior to these images being taken with a history of acquired tendon contracture, a stiff stilted gait, angular limb deformities and poor growth. This foal was owned by a desert Bedouin and fed a diet of barley and locally grown rhodes hay. A periosteal elevation was performed on the right knee (the physis was still open) and the foal was bandaged and box rested in addition to dietary alterations being made. Note the grossly evident expansion of the carpal and metacarpophalangeal physes. This foal had a PTH level of 52 pg/mL.

- Signs such as facial alterations although described as the classical lesions are rarely seen in foals with the condition.
- Pain and stiffness when moving (related to expansion of growth plates) may be evident before there are significant radiographic changes and is the most common presentation seen by the author.



Figures 13.4 & 13.5: Dorsopalmar metacarpophalangeal radiographs from the foal in Figs 13.2 & 13.3 (on the left) and from an age-matched control (on the right). Note the expansion and delayed closure of the physis in the foal with hyperparathyroidism when compared to the age-matched control.

- Angular limb deformities as a result of changes in growth pattern of the limbs.
- Pathological fractures are rare but can occur.

Diagnosis

- Radiographic loss of bone density may be helpful but is not diagnostic and can be hard to critically assess.
- Slow closure and expansion of physes in foals with a suggestive dietary history should be regarded as highly suspicious and should have PTH measurements performed.
- Serum calcium and phosphorus levels are normal but urinary levels of phosphorus may be increased.
- Immunoassays for measurement of PTH are commercially available. Immunoradiometric assays for measurement of human intact PTH have also been validated for use in the horse. Normal valves are <40 pg/mL.

Treatment and prognosis

- Rest and dietary adjustment of Ca : P ratio.
- Obvious deformities may require specific treatment.
- Pathological fractures are rarely treated.
- The prognosis is dependent on how advanced the disease process and associated bony alterations had become before detected. Anatomical distortions are often irreversible.

Metabolic disorders

Hypoglycemia

History

Newborns have limited energy reserves and any factor that limits energy intake can result in profound hypoglycemia. Hypoglycemia also commonly occurs with generalized infections and is associated with bacterial consumption and reduced glycogen reserves. Foals that are regarded at increased risk:

- premature or small for gestational age
- cesarian section or dystocia with anesthesia
- neonatal isoerythrolysis foals
- hypothermia
- asphyxia and hypoxia
- septicemia
- hypoxic ischemic encephalopathy
- inherited metabolic defects (lysosomal storage disease)
- previous bolus injections of glucose or rapid rate of glucose infusion and sudden cessation results in rebound hypoglycemia, such as when parenteral nutrition is abruptly discontinued
- orphan foals
- liver failure (Tyzzer's disease)
- hyperlipidemia.

Clinical signs

Clinical signs are variable and can include lethargy, bradycardia, inability to rise, coma or seizures. In foals with suspected underlying conditions such as HIE early correction of hypoglycemia is important to determine the extent to which the hypoglycemia has contributed to the overall appearance. Many foals improve dramatically after correction of hypoglycemia.

Hypoglycemic foals can be split into two categories:

- Asymptomatic. No detectable clinical signs. Hypoglycemia is detected on blood analysis and is defined as a blood glucose <40 mg/dL in a presuckle foal and <80 mg/dL in a foal older than 2 hours which has suckled.
- Symptomatic. Foals in which any of the above clinical signs are present that disappear with glucose administration regardless of the blood glucose level.

Diagnosis

- Determination of blood or serum glucose is an important part of the assessment of any abnormal foal.
- Whole blood glucose levels can be rapidly determined at farm level by the use of reagent strips and glucometers. Glucometers designed for use by human diabetic patients are easy to use, give relatively reliable results and are extremely cost effective when used for serial monitoring (see Fig 3.67).
- A delay in separation of red cells from serum can result in a spuriously low glucose as a result of metabolism of glucose by the cells.

Treatment

- *Administer 5–10% dextrose solution at 4–8 mg/kg/min. In a 50 kg foal, 120–240 mL of 10% dextrose would be infused per hour.*
- Severe hypoglycemia can be treated with an initial more rapid infusion (5–10 mL/kg of 10% dextrose rapidly) and then should be followed by a slower continuous infusion.
- Re-check glucose level in one hour and monitor every 4–6 hours thereafter.
- Begin feeding if possible (no reflux) – either orally or by nasogastric tube.
- Correct any other predisposing causes, e.g. hypothermia, acid–base imbalances or hypoxemia.
- Repeated boluses of hypertonic glucose should not be given instead of a continuous infusion as it may aggravate pre-existing CNS damage, result in rebound hypoglycemia and is irritating to veins.
- Hyperglycemia (>180 mg/dL) should be avoided. If noted then stop the glucose infusion and re-evaluate the blood glucose levels at hourly intervals until normalized. Once normalized decrease the strength of the glucose infusion by 50% or discontinue the glucose infusion.
- It should be noted that blood glucose may be successfully maintained with an infusion rate of 5 mg/kg but this rate of infusion will only provide 24 kcal/kg/day, which is well below the foal's nutritional requirements.

Malnutrition (Figs 13.6 & 13.7)

History and etiology

Poor growth rates and/or weight loss in foals can have many causes and is a common occurrence. Frequently the changes may be subtle and are not immediately noticed by the owner. This can be compounded by the fact that many weight assessments on newborn foals are done subjectively. Such foals are often presented for "not doing right" and after extensive and often expensive testing has been performed malnutrition may be diagnosed. Milk provides the majority of the foal's needs in the first 12 weeks of life and thus anything that decreases milk supply will directly affect the foal. This can be a somewhat easy diagnosis in a mare that is obviously in poor condition. It can be somewhat more challenging in a mare which appears to be in good condition with a foal which is not thriving.

Diagnosis

The approach to the foal which is demonstrating a poor growth rate and/or weight loss should start with a thorough clinical examination which may reveal an underlying cause or a clue to that cause, e.g. heart murmur associated with congenital cardiac defect, fever or injected mucous membranes associated with septicemia. If the initial examination indicates a healthy foal then assessment of the mare's milk supply should be made:

- A foal which is seen to make very frequent attempts to nurse and get frustrated while doing so may be an indication of an inadequate milk supply.
- Muzzling the foal for 2–3 hours should result in an engorged udder in a mare with normal milk production. An udder that remains soft after muzzling indicates inadequate milk production.

Mismothering should also be considered in a mare which appears to have adequate milk production. It is important to note that some mares will only show aggressive tendencies towards their foals when no-one is watching. Such cases may require video monitoring.

If milk production and mismothering have been ruled out then other testing (blood work, cardiac ultrasound, gastroscopy) should be performed.

Differential diagnosis

0–7 days

- Malnutrition
- Mismothering
- Poor management, low ambient temperature
- Infections:
  - septicemia
  - umbilical infection
- Congenital defects
- Cardiac defects – severe defects would have to be present to demonstrate poor growth at this stage
- Dental deformity – again severe defects would have to be present at this stage, as most foals will continue to nurse and will only show signs later on when a greater percentage of their diet is roughage

Figures 13.6 & 13.7: Malnutrition. This is a 3 day old foal that was only allowed to nurse by the mare when she was being observed. At the time this image was taken he was 6Kgs lighter than his birth weight. Note also the bite wound at the level of the foal's withers inflicted by the mare.

- Hypothyroidism – usually associated with marked clinical signs
- Miscellaneous causes:
  - gastroduodenal ulceration – uncommon in otherwise healthy foals in this age group
  - immunocompromised.

After the first week

In addition to the above:

- Chronic infections such as *R. equi*
- Parasitism
- *Lawsonia intracellularis*

Treatment

If poor milk production is determined to be the cause then several approaches can be taken:

- If the mare is recently foaled (1–3 days) and especially if a maiden mare is involved, a conservative approach of monitoring the mare's milk production may be taken as some mares require a number of days to come into a good milk supply. This should be combined with providing supplemental nutrition to the foal either from a bucket or by nasogastric tube. Most foals that have already nursed from the mare's udder will not accept a bottle.
- The above method can be combined with an attempt to increase the mare's milk production by administration of domperidone (1.1 mg/kg orally q12–24h to effect).
- If the foal is already a number of weeks old or a conservative approach has not worked then it may be necessary to either continue offering supplemental milk from a bucket or to foster the foal. Hand rearing should be avoided whenever possible as social adaptation of such foals can be difficult.

Recommended reading

Bertone J J 1992 Nutritional secondary hyperparathyroidism. In: Robinson NE (ed) Current therapy in equine medicine, 3rd edn. WB Saunders, Philadelphia, p 119–122

Beyer MJ, Freestone JF, Reimer JM et al 1997 Idiopathic hypocalcemia in foals. Journal of Veterinary Internal Medicine 11(6):356–360

Little D, Redding WR, Spaulding KA et al 2000 Unusual presentation of nutritional secondary hyperparathyroidism in a paint colt. Equine Veterinary Education 12(6):297–302

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Taribio RE 2004 The adrenal glands. In: Reed SM, Bayly WM, Sellon DC (eds) Equine internal medicine. WB Saunders, St Louis, p 1357–1361

CHAPTER 14

The eye and related structures

Nathan M Slovis DVM, DipACVIM, CHT

Ophthalmologic examination of the foal (Figs 14.1–14.5)

Ophthalmic examination needs to be systematic and complete. Establish a routine method of examination of the whole eye, lids, conjunctiva, cornea, anterior chamber, iris, lens, vitreous, retina and globe. Use of an eye examination sheet with pictures or photographs allows for comparison in follow-up examinations. A thorough history is important and should include the following questions:

- How long has the problem been present?
- Has it progressed?
- What medications have been given and was any improvement noted with medication?
- Are one or both eyes affected?
- Is the vision better at night?
- Do any other members of the family have or have they had eye conditions?

Steps in an ophthalmic examination

1. Symmetry of the orbits, sinuses and globe position should be evaluated.
2. Eyelids are evaluated for symmetry, entropion, lacerations and blepharospasm. Severe eyelid swelling may be associated with blunt trauma and/or corneal ulceration.
3. Both the cornea and the pupil are oval horizontally. A pigmented ring encircles the limbus spreading 1–2 mm into the cornea. At the superior border and, to a lesser extent, the inferior border of the iris there are pigmented bodies that are thought to aid in decreasing glare. These pigmented bodies have been referred to as either granula iridica or corpora nigra. They are vascularized cystic protrusions of the posterior iridal pigmented epithelium.
4. Pupillary light reflex (PLR). This reflex evaluates the afferent function of the retina, optic nerve, optic tracts, as well as the efferent function of the oculomotor nerve and iris sphincter muscle function.
   - While evaluating the PLR one should evaluate pupil symmetry. Using the direct ophthalmoscope at 0 diopters to observe the tapetal reflections of both eyes, while standing 6 feet directly in front of the foal, will allow one to easily observe differences in pupil size (anisocoria).
   - Direct PLR refers to the degree of constriction that is noted when a bright light is directed into the ipsilateral pupil.
   - Consensual PLR is when the contralateral pupil also constricts during the direct PLR.
   - *Remember* that a normal PLR does not confirm vision because vision is a cortical phenomenon, not a reflex.
5. Menace response. The visual pathway is tested by the menace response. The hand is moved rapidly toward the eye in a threatening gesture – this results in eyelid closure and the head may be jerked away.
   - For practical purposes the vision in one eye is perceived by the visual cortex of the contralateral cerebral cortex as there is 80–90% crossing of optic fibers at the optic chiasm.
   - The motor component is mediated through the facial nerve and its nucleus is in the medulla.
   - The afferent side of the response is extensive and involves a cerebral pathway, which implicates a learned response. It is usually present by 5–10 days in foals. Therefore testing the menace response in a neonatal foal will be unrewarding (see Fig 3.47).

Figure 14.1: Auriculopalpebral nerve block. The nerve runs over the highest point of the zygomatic arch and can be palpated after dampening the area with alcohol. 2 mL of local anesthesia is used and results in akinesia of the eyelids in 5–10 minutes.

Figure 14.2: Supraorbital block (frontal branch of the trigeminal nerve). 4 mL of local anesthetic is infused into the supraorbital foramen. This block is not normally required for examination of the eye but is useful for the placement of subpalpebral lavage systems.

Figure 14.3: Topical block of the cornea using lidocaine. The needle is removed from the hub to allow the solution to be sprayed into the eye.

Figure 14.4: Scraping the corneal surface with the blunt end of a scalpel blade to obtain a cytological sample.

- Some animals may not respond to a menace response test and a true visual deficiency may be detected by placing objects in front of the animal and observing its behavior – or ideally by performing a maze test.
- Menace deficit can also be the result of facial nerve paralysis in which case the animal is unable to blink, but usually will show avoidance movements of the head. Such animals will also have other signs of facial nerve involvement, e.g. facial drooping on the same side.
- Animals with cerebellar disease may also display a menace deficit, yet possess normal vision. The precise mechanism by which the cerebellum effects this pathway is not known but is thought to involve upper motor neuron control of the facial nerve. These animals retain visual acuity and will be able to perform a maze test.

6. Sedation and an auriculopalpebral nerve block can then be performed. They should be avoided prior to evaluation of the PLRs and menace response as they may alter the results of these examinations.
7. Microbiological samples are ideally taken prior to the administration of topical anesthesia. In certain fractious individuals it may be necessary to sterilely apply topical anesthesia prior to sampling. Cytological samples can be obtained after the application of topical anesthesia.
8. Fluorescein staining can then be performed to evaluate corneal integrity.
9. The eyelids including the third eyelid can then be everted and examined.
10. Intraocular pressure (IOP) can then be measured – if normal (20–25 mmHg) the pupil can be dilated. A tonometer should be

Figure 14.5: Fluorescein staining of the new born foal's eye. The syringe is filled with 2 mL of sterile lidocaine with fluorescein stain strip immersed in the lidocaine for 3 seconds and then removed. Again the needle is removed from the hub to allow the solution to be sprayed into the eye.

Figures 14.7: Congenital cataract in a 3-week-old TB foal. The foal had bilateral cataracts and was bilaterally blind.

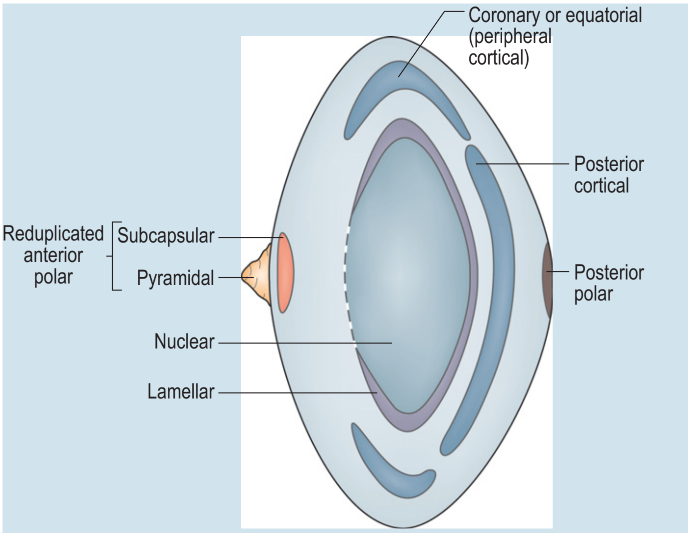


Figure 14.6: Classification of cataracts according to position within the lens.

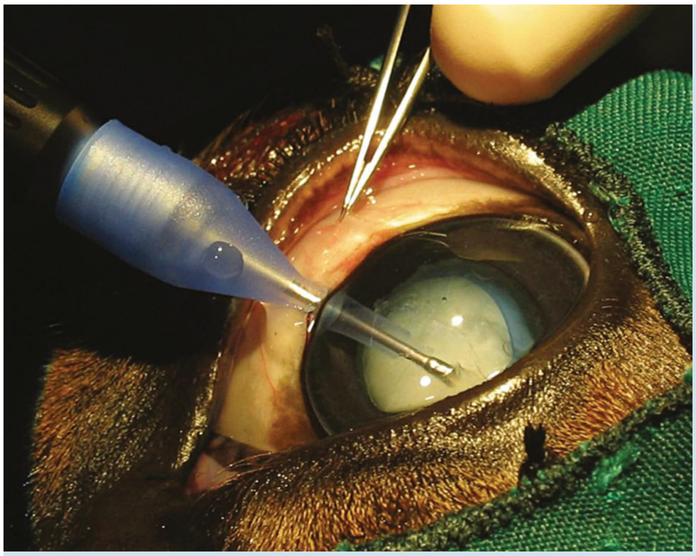


Figure 14.8: Phacoemulsification of the lens in Fig 14.3. The foal was noted to be healthy, with no history of uveitis or other ocular problems. Early return to vision is necessary for the development of higher visual centers. This foal was an ideal surgical candidate because of not only the animal's ocular health but of his personality to allow proper topical therapy postoperatively. This procedure was accomplished through a small limbal incision utilizing a piezoelectric handpiece with a titanium needle in a silicone sleeve to ultrasonically fragment and emulsify the lens nucleus and cortex.

used for measuring IOP and should be part of the equipment of any practice that receives eye cases.

11. Following dilation of the pupil the anterior structures can be examined followed by the lens, vitreous and fundus.
12. The nasolacrimal duct may then be irrigated if required.

Congenital anomalies

Cataracts (Figs 14.6–14.9)

Cataract is the name given to an opacity of the lens that is the most common ocular anomaly in foals. Cataracts may be associated with other congenital eye defects such as microphthalmos, persistent pupillary membranes, persistent hyaloid structures or multiple ocular anomalies.

Cataracts represent 33.6–35.3% of congenital ocular defects recorded. A cataract is described by its location within the lens. Capsular, subcapsular, cortical, and nuclear cataracts may be further localized to anterior, posterior, polar or axial, and equatorial. Most congenital cataracts are nuclear. Cataracts can further be described on the basis of severity from incipient, immature and mature, to hypermature and Morgagnian.

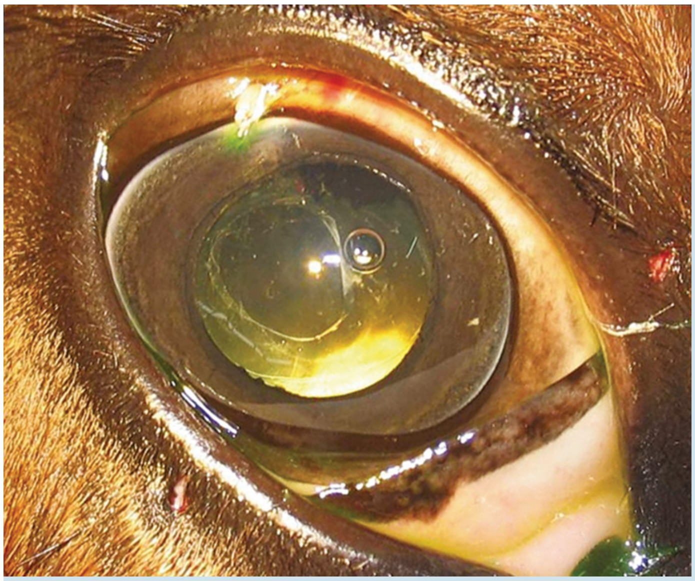


Figure 14.9: Successful removal of the cataract. Results of cataract surgery are generally good in foals. Most foals have to continue medications for up to 2 months. This foal was started on bacitracin, neomycin, polymyxin B with dexamethasone 4× daily, topical atropine ointment 2–4× daily until the pupil was dilated and then as needed, topical diclofenac 4× daily for 1 week, flunixin meglumine 1 mg/kg IV q 12 hours for 1 week and then orally 1× daily for another week, and omeprazole 2 mg/kg PO SID for 28 days. All medications were stopped at 4 weeks postoperatively.

Congenital cataracts may be present at birth or may take up to 3 months to become apparent. Congenital cataracts may occur as a result of an insult to the developing fetus in the form of an infectious, toxic, traumatic or other insult. Cataracts may also be inherited, with a dominant inheritance pattern reported in Belgian and Thoroughbred horses.

Morgan horses have a non-progressive, nuclear, bilaterally symmetrical cataract that does not generally interfere with vision. The Rocky Mountain Horse has a heritable eye disease, anterior segment dysgenesis, which is characterized as having cataracts and lens luxation.

Clinical signs

- No clinical signs may be evident.
- Visual impairment may be demonstrated by reluctance to run or hitting objects in the stall/environment.
- Opacity of the lens may be obvious.
- The presence of other eye abnormalities warrants a closer examination of the lens as there is a high incidence of cataracts in foals with other congenital eye anomalies.
- Abnormal head posture may be associated with unilateral cataracts because the foal would turn the head towards the intact sighted eye.

Diagnosis

- Examination of the eye with an ophthalmoscope or other transilluminator such as a penlight may be all that is required for a diagnosis. It is however important to assess the extent of the cataract by using a mydriatic agent (e.g. tropicamide 2%) to dilate the pupil fully.
    - ♦ Focal dense nuclear cataracts appear as a series of hollow rings or a sphere. Foals with these cataracts often have much improved vision following mydriasis and may appear to see better in low light conditions which cause pupillary dilation. In some of these foals the cataract may appear to get smaller over time as the lens and related structures increase in size while the cataract remains the same.
    - ♦ Diffuse mature cataracts have a more uniform opacity and result in blindness in the affected eye.
- Ultrasonographic examination of the eye is important for further characterizing the cataract but also allows examination of other structures of the eye, abnormalities of which may have a significant bearing on the overall prognosis.

Differential diagnosis

- Acquired cataract resulting from trauma, ocular infection and inflammation. It is important to try to differentiate between congenital and acquired cataracts as there is often a much poorer prognosis associated with acquired cataracts.
- Corneal opacity resulting from such conditions as ulceration or entropion.
- Other causes of central or ophthalmic blindness such as congenital abnormalities or trauma.

Treatment

Congenital nuclear cataracts can be removed successfully. The preferred method is by phacoemulsification. It has been suggested that foals should be screened for subclinical *Rhodococcus equi* and *Streptococcus* pneumonia prior to removal as sight-threatening postoperative endophthalmitis may result in 7–10 days following surgery if these conditions are not treated before cataract surgery.

It has also been suggested that there is a latent period in which treatment is likely to result in vision in foals with bilateral dense congenital cataracts. The exact time frame of this latent period is not known but treatment should be performed prior to the onset of nystagmus. In human infants with similar conditions the latent period ends with the onset of nystagmus at 10 weeks.

Ankyloblepharon / symblepharon (Fig 14.10)

Failure of the eyelid to separate (ankyloblepharon) or adhesion of the eyelids to the cornea (symblepharon) occurs rarely in foals. Ankyloblepharon may be seen in premature foals while symblepharon is associated with intrauterine corneal or conjunctival inflammation.

Diagnosis

- Failure of the eyelids to separate and open following birth is usually noted by the owner.
- Ultrasound of the globe can help rule out other ocular abnormalities before the eyelids are opened. These conditions may occur alone with a normal eye beneath the closed eyelid or can occur as part of a complex of ocular anomalies.

Figure 14.10: Ankyloblepharon in a Standardbred colt. The eyelids were manually separated from each other.

Figure 14.11: Dermoid.

Treatment

- For ankyloblepharon the eyelids are gently separated from each other manually and surgical intervention is usually not needed.
- Symblepharon usually requires surgical intervention in order to preserve the underlying structures.

Ocular dermoid (Fig 14.11)

Ocular dermoids are congenital masses of normal tissue arising on the palpebral or bulbar conjunctiva or the third eyelid. In rare cases the limbal margin of the cornea may be affected. Dermoids contain many of the elements of normal skin – epidermis, dermis, fat, sebaceous glands and hair follicles.

Diagnosis is made by simple observation. This condition occurs alone and is not associated with other ocular abnormalities.

Treatment

Surgical removal is curative. Some cases that involve the cornea may require a keratectomy.

Anophthalmia / microphthalmia (Figs 14.12 & 14.13)

Microphthalmia refers to an abnormally small eye. This congenital abnormality is noted especially in Thoroughbreds and can affect one or both eyes. Sight in the affected eye may be normal. A greater than 10% reduction in globe size is possibly associated with sight deficits. Severely affected eyes are usually blind.

Anophthalmia is a rare event and these show absence of any eye structure, which *may* or *may not* include adnexal structures.

These conditions are not thought to be inherited but rather result from a toxic or infectious insult in utero and as such should not mitigate against continued breeding from the parents.

Figure 14.12: Microphthalmia in a Thoroughbred colt. Note the smaller than normal globe. This eye was not visual. The animal had bilateral microphthalmia.

Distichiasis / trichiasis (Figs 14.14 & 14.15)

Distichiasis is a disorder of the eyelashes (cilia) in which additional cilia emerge from the openings of the tarsal glands resulting in abnormal contact with the cornea. Trichiasis involves outer cilia and adjacent skin hairs pointing in an abnormal direction toward the cornea. Both conditions are rare.

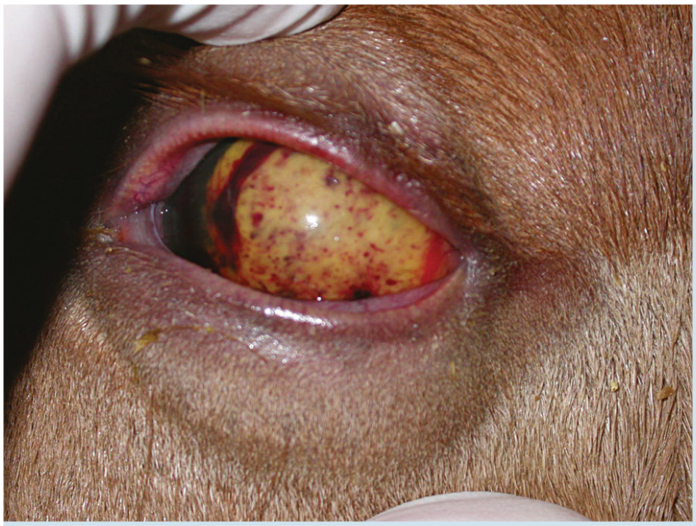


Figure 14.13: Rare hepatoblastoma that metastasized to the cornea in a newborn foal. This is not to be confused with microphthalmia.

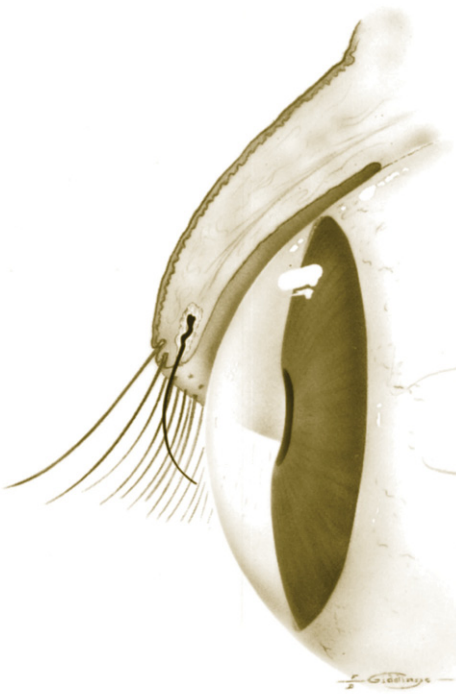


Figure 14.14: Distichiasis (from Slatter D 2001 Fundamentals of Veterinary Ophthalmology, 3rd edn. WB Saunders, St Louis).

Clinical signs

Corneal ulceration with accompanying corneal edema and lacrimation.

Diagnosis

A simple visual inspection may reveal the abnormal hairs but usually the use of a magnifying aid is required.

Figure 14.15: Trichiasis (from Slatter D 2001 Fundamentals of Veterinary Ophthalmology, 3rd edn. WB Saunders, St Louis).

Treatment

Treatment consists of surgical removal of the abnormal single or multiple hairs. Electrolytic depilation may result in long remission but may not be permanent.

Persistent hyaloid artery (Fig 14.16)

The hyaloid artery arises from the primitive ophthalmic artery stretching from the optic disk to the lens and enveloping the posterior lens capsule. The artery appears ophthalmoscopically as a red or white spot anterior to the optic disk when viewed end-on and extends a variable distance into the vitreous. It provides nutrition to the developing lens.

Remnants of this embryonic vasculature are present in 80% of neonatal Thoroughbred foals that are examined within 48 hours of birth. The hyaloid artery usually atrophies within a few days but a large variation in time can be expected.

The remnants have little effect on the horse's future vision and are more important in a differential diagnosis than as a cause of detrimental visual effects.

Persistent pupillary membranes

Remnants of the fetal blood supply to the lens arise from the iris collarette. These strands may arise freely into the aqueous or attach to the lens or endothelium of the cornea. At times the strands may span across the iris face from collarette to collarette. Anterior capsular cataract can result from the membranes attaching to the anterior lens

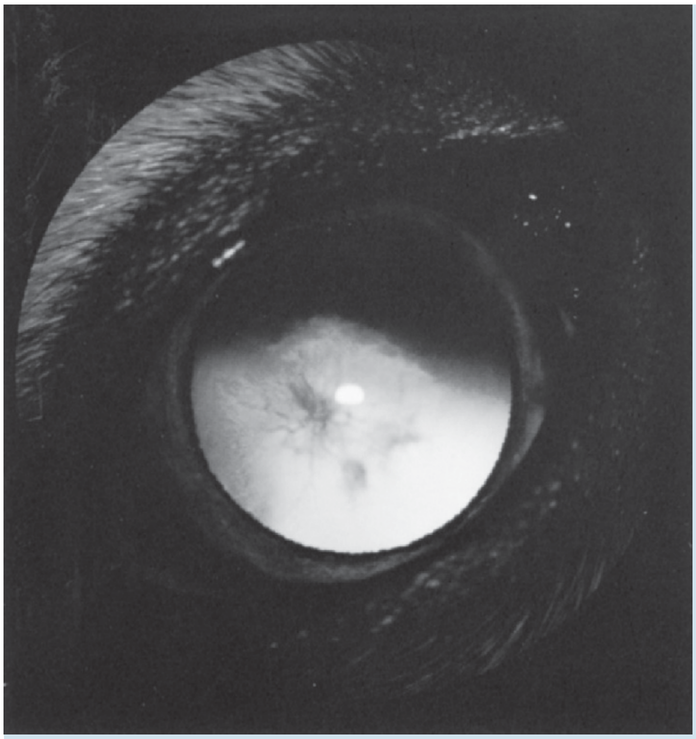


Figure 14.16: Persistent hyaloid artery (from Slatter D 2001 Fundamentals of Veterinary Ophthalmology, 3rd edn. WB Saunders, St Louis).

Figures 14.17 & 14.18: Entropion. Inward rolling of the eyelid margin. Note the uveitis indicated by the green hue to the iris and fibrin accumulation in the anterior chamber.

capsule. Strands attaching to the cornea can cause a leukoma that may cause some visual deficits.

No treatment is usually warranted.

Entropion (Figs 14.17–14.21)

Entropion is the inward rolling or turning of the eyelid margin. The lower eyelid is much more commonly affected. Neonatal entropion may occur as a primary defect and is inherited in the Thoroughbred. Secondary entropion tends to occur with either dehydration or spasm of the orbicularis oculi muscle following a painful ocular disorder. Dehydration tends to cause foals to be enophthalmic (loss of periorbital tissue/fat), and this will allow the eyelid to roll in. This enophthalmia is commonly seen in the sick neonate.

Clinical signs

- Clinical signs may include blepharospasm, excessive lacrimation, epiphora, corneal ulceration and edema.
- ***Corneal sensitivity in the newborn is poor and there may be no pain associated with this condition.*** Careful examination is therefore warranted for every newborn foal at the first routine examination. Foaling staff should also be trained to recognize the condition.

Treatment

Treatment depends on the cause of the entropion.

- In dehydrated foals intravenous fluids along with ocular lubricants and manual eversion of the affected eyelid several times a day while the animal becomes rehydrated may be all that is necessary.
- The use of either procaine penicillin (0.5–1 mL) injected into the affected eyelid's subcutaneous tissue or squeezing the affected eyelid firmly between the thumb and forefinger along its entire length will induce subcutaneous edema. The eyelid may then be manually everted and often remains in place.
- If surgery is required then three or four horizontal mattress sutures may be necessary. Lidocaine injected into the subcutaneous tissue of the affected eyelid and/or sedatives (diazepam 0.1 mg/kg IV) may have to be used in foals that are alert and moving during the procedure.

Coloboma

A coloboma is a defect in one or more of the structures of the eye or the adnexa. They occur in the eyelids, iris, retina and the optic disc.

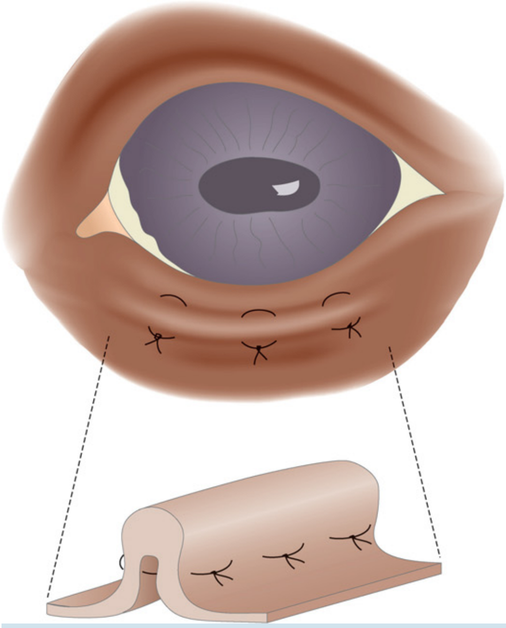


Figure 14.19: (a) Entropion sutures. Primary entropion in the foal is simple to treat with carefully placed vertical mattress sutures in the lower eyelid to reduce the extent of the turning-in. The sutures are not tied until all are laid so that the exact tension can be used to cure the problem. Surgical excision of the skin should be avoided as far as possible. (b) Entropion surgery resulting in restoration of eyelid contact without skin removal (from Knottenbelt DC et al 2004 Equine neonatal medicine and surgery. Saunders, Philadelphia).

Figure 14.20: Horizontal mattress sutures to fix entropion. The sutures will be removed in 5–7 days.

Figure 14.21: Entropion fixed with the injection of 0.5 mL of procaine penicillin into the subcutaneous tissue of the lower eyelid.

Some iris colobomas are inherited. Coloboma may occur alone or with other ocular anomalies.

Clinical signs and diagnosis

- Eyelid colobomas are the most apparent and present as obvious non-inflammatory defects. Agenesis of the eyelids is rare and has been reported in only one foal. Short full-thickness defects are more common and occur most frequently on the upper eyelid.
- Other cases of eyelid colobomas may include a small notch in the eyelid which requires little or no treatment.
- Eyelid coloboma may be associated with trichiasis causing corneal irritation; cryoepilation may be required.
- Internal colobomas frequently go unnoticed as they require a more thorough ophthalmic examination for detection.

Figure 14.22: Iris hypoplasia (from Knottenbelt DC et al 2004 Equine neonatal medicine and surgery. Saunders, Philadelphia).

Treatment and prognosis

- The treatment chosen will depend on the extent of the lesion. Small lesions may require no treatment.
- Larger lesions can be difficult to treat. The initial main objective is to prevent corneal drying. Immediate care may be provided by either a contact placement or ocular lubricant. It may be possible to reconstruct an eyelid even though this may have little muscular function.
- There is no treatment for intraocular coloboma.
- The prognosis is usually good with most affected eyes having normal vision. The size of an eyelid coloboma determines its prognosis with larger lesions having implications for eyelid function. Theoretically optic disc coloboma should have a profound effect on vision but the incidence is so low that it is poorly documented. The presence of other ocular abnormalities may alter the prognosis.

Iris abnormalities (Fig 14.22)

True absence of the iris is rare in horses. Iris hypoplasia has been noted in normal horses of varying ages and breeds. The superior iridial region appears to bulge toward the cornea and apparently is more common in blue irides.

Atresia of the nasolacrimal duct (Figs 14.23–14.26)

Complete atresia of the nasolacrimal duct is rare, as is absence of the palpebral puncti. Absence of the nasal punctum is the most common presentation with or without atresia of the distal portion of the duct. The condition can be bilateral but more commonly is unilateral.

Clinical signs

- Most cases present with a mucopurulent ocular discharge at 2–4 months of age. Ocular secretions accumulate within the affected duct over time and a secondary dacryocystitis develops.
- Lacrimation epiphora may be seen but in many cases goes unnoticed for months as foals have a lower tear volume than adults.
- Facial excoriation may be seen in some foals.

Diagnosis

- Direct examination usually reveals an absence of a nasal punctum. The punctum is usually present at the junction of pigmented and non-pigmented mucosa. It may also be possible to visualize the distended distal end of the duct. Light pressure applied to the duct may cause a backflow in the duct with increased ocular discharge.
- Placing a small amount of fluorescein solution in an eye should result in stain being apparent at the ipsilateral nostril within a few minutes. Thus absence of staining when a nasal punctum is present indicates atresia or blockage along the length of the duct.
- Retrograde placing of a catheter through the palpebral punctum should be performed to determine the level at which the duct is atretic or blocked.
- Dacryocystorhinography is indicated when the simpler tests outlined above fail to yield a positive diagnosis but is rarely required.

Treatment

- A nasal punctum is created surgically by making a 3–4 mm incision with a #15 scalpel blade over a polyurethane catheter (dog urinary catheter works well) placed in the duct via the palpebral punctum.
- It is possible to perform the procedure standing using topical ophthalmic anesthesia and chemical restraint. However, the placement of the catheter in the palpebral punctum may be difficult in some horses and for this reason general anesthesia is preferred.
- When the catheter tip has been exposed it is threaded through the false nostril and either sutured to the side of the face or the catheter ends tied together and taped to the face. The catheter is left in place for 14–21 days, until epithelialization is complete.
- Electrocoagulation of the incision site or attempts to suture the duct lining to the mucosa of the nostril should be avoided as both result in stricture formation.
- If the duct is atretic along its length, dacryoconjunctivorhinostomy in the malar region may be required to provide permanent drainage of the duct system.

Acquired diseases

Corneal ulceration (Figs 14.27–14.45)

There is evidence that corneal sensitivity in foals is very poor. This coupled with lower tear production in foals compared to adult's results in an increased incidence of corneal disease in sick neonatal foals.

Figure 14.23: This 3-month-old foal with bilateral atresia of the nasal puncta of the nasolacrimal duct presented with bilateral mucopurulent ocular discharge.

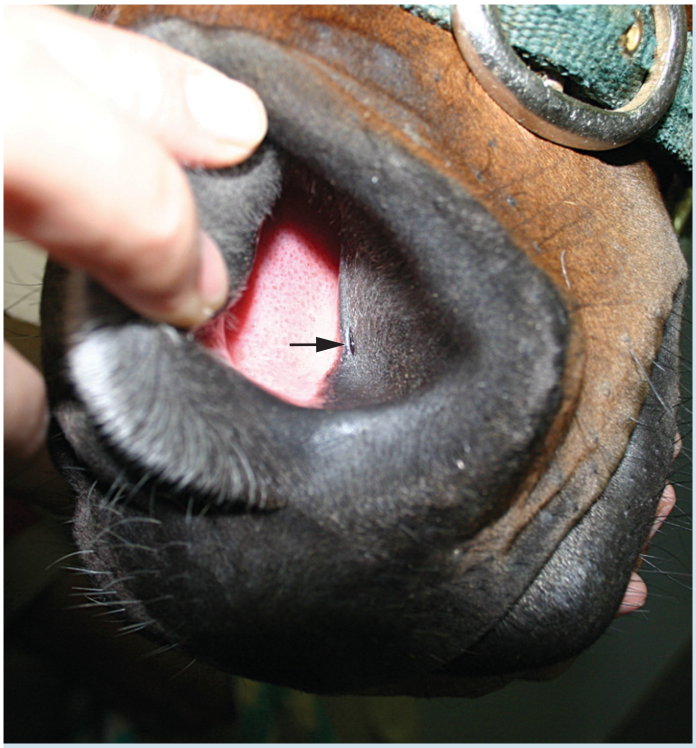


Figure 14.24: Normal nasal punctum of the nasolacrimal duct. The punctum is usually located at or close to the junction of the pigmented and non-pigmented mucosa (arrow).

Figure 14.25: Same foal as Fig 14.23; note the absence of the nasal punctum (compare with Fig 14.24).

Figure 14.26: A dog urinary catheter was advanced along the nasolacrimal duct from the ocular puncta. Note the bulging of the mucosa (arrow) where the duct ends.

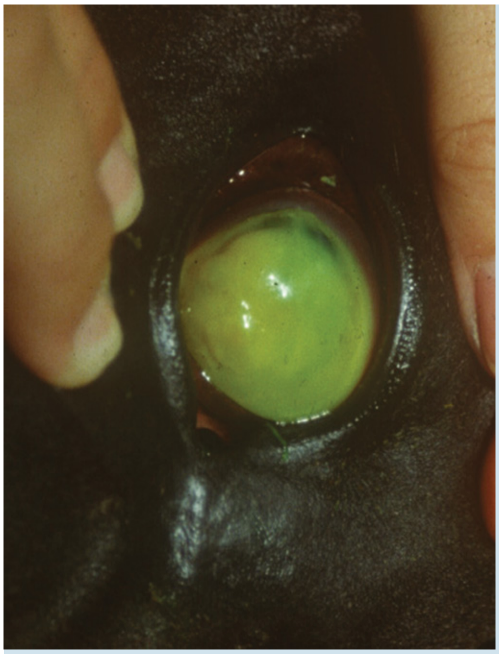


Figure 14.27: A severe corneal ulceration secondary to foreign debris (dirt) that was in this recumbent foal's eye. Routine cultures failed to reveal any bacterial growth. The foal was maintained on neomycin, bacitracin and polymyxin B ointment and 1× daily atropine ointment for 2 weeks until healing was complete.

Figure 14.28: A 4-month-old TB foal presented for complaint of excessive ocular discharge and marked blepharospasm of the right eye. The owner stated that the eye was of normal appearance the day before admission.

Figure 14.29: The foal in Fig 14.28 was sedated with 100 mg of xylazine IV and had an auriculopalpebral nerve block performed. Marked corneal edema, keratomalacia and stromal infiltrates were noted. The eye was flushed with 2% lidocaine and sterile saline.

Figure 14.30: A fluorescein stain was performed on the foal in Figs 14.28 & 14.29 and revealed a 1 cm × 1 cm 50% depth central corneal ulcer. Culture isolated *Streptococcus zooepidemicus*.

Figure 14.31: Granulation tissue bed is apparent at the site of the old corneal ulceration (same foal as Figs 14.28–14.30, 7 weeks after presentation). Treatment in this foal consisted of the placement of a subpalpebral lavage system. Rotating topical application of chloramphenicol 1% 0.2 cc q2h and neomycin, polymyxin B and bacitracin (Neopolybac ophthalmic^{R}) 0.2 cc q2h, such that an antibiotic was being administered every hour. Topical ophthalmic atropine 1% 0.2 cc q6h, serum 0.2 cc q4h and parenteral flunixin meglumine 1 mg/kg IV q12h. After 2 weeks the subpalpebral lavage system was removed and the animal was changed to chloramphenicol ophthalmic ointment and Neopolybag rotating q4h, atropine 2× weekly and the flunixin meglumine was discontinued.

Figure 14.32: There are various lavage systems present in the veterinary market. A commonly used system is the one-hole subpalpebral lavage system (Mila International Inc., Florence, Kentucky, USA) which is placed in the superior lid.

Figure 14.33: Side-on view of the end plate of the one-hole subpalpebral lavage system.

Extensive ulceration may be present with minimal blepharospasm and epiphora. Determination of corneal integrity and routine fluorescein staining should be a part of every ophthalmic system examination.

The cornea is a five-layered membrane of which the stroma forms the bulk. The stroma is bordered outwardly by the corneal epithelium and its basement membrane. Inwardly the stroma is bordered by the Descemet's membrane. Descemet's membrane does not stain with fluorescein, and it appears as a dark transparent, outwardly bulging structure in the center of a deep corneal ulcer or wound that is about to rupture. The endothelium is one cell thick and lies posterior to Descemet's membrane. The endothelial cells actively pump fluid out of the cornea, thus allowing the normal state of dehydration necessary for corneal clarity. The cornea inwardly is bathed by the hypertonic aqueous humor and externally by hypertonic tears. The effects of these two hypertonic fluids are to maintain the cornea in a deturgescent state by pulling out excessive water.

Diagnosis

- All inflamed, painful or cloudy eyes should be routinely stained with fluorescein. The fluorescein stain allows evaluation of the cornea's integrity because the dye is hydrophilic, binding the corneal stroma when ulcers are present. The fluorescein enters the water-soluble corneal stroma and stains the intracellular spaces. The dye will not bind to intact healthy corneal epithelium or Descemet's membrane. It may be easier to immerse the paper strip in 1 mL of sterile saline or lidocaine in a 3 mL syringe with the needle removed from the hub and use this to gently squirt the cornea.
- Examination of cytological samples and microbiologic cultures of rapidly progressing or deep ulcers are extremely useful.
  - Cytological samples may be obtained with the use of the blunt end of a scalpel blade or the tip of a sterile moistened cotton swab and topical anesthesia (see Fig 14.4).
  - Cultures should be obtained with a sterile moistened cotton swab before administration of topical antibiotic/antifungal solutions or ointments.

Superficial erosions (scratch)

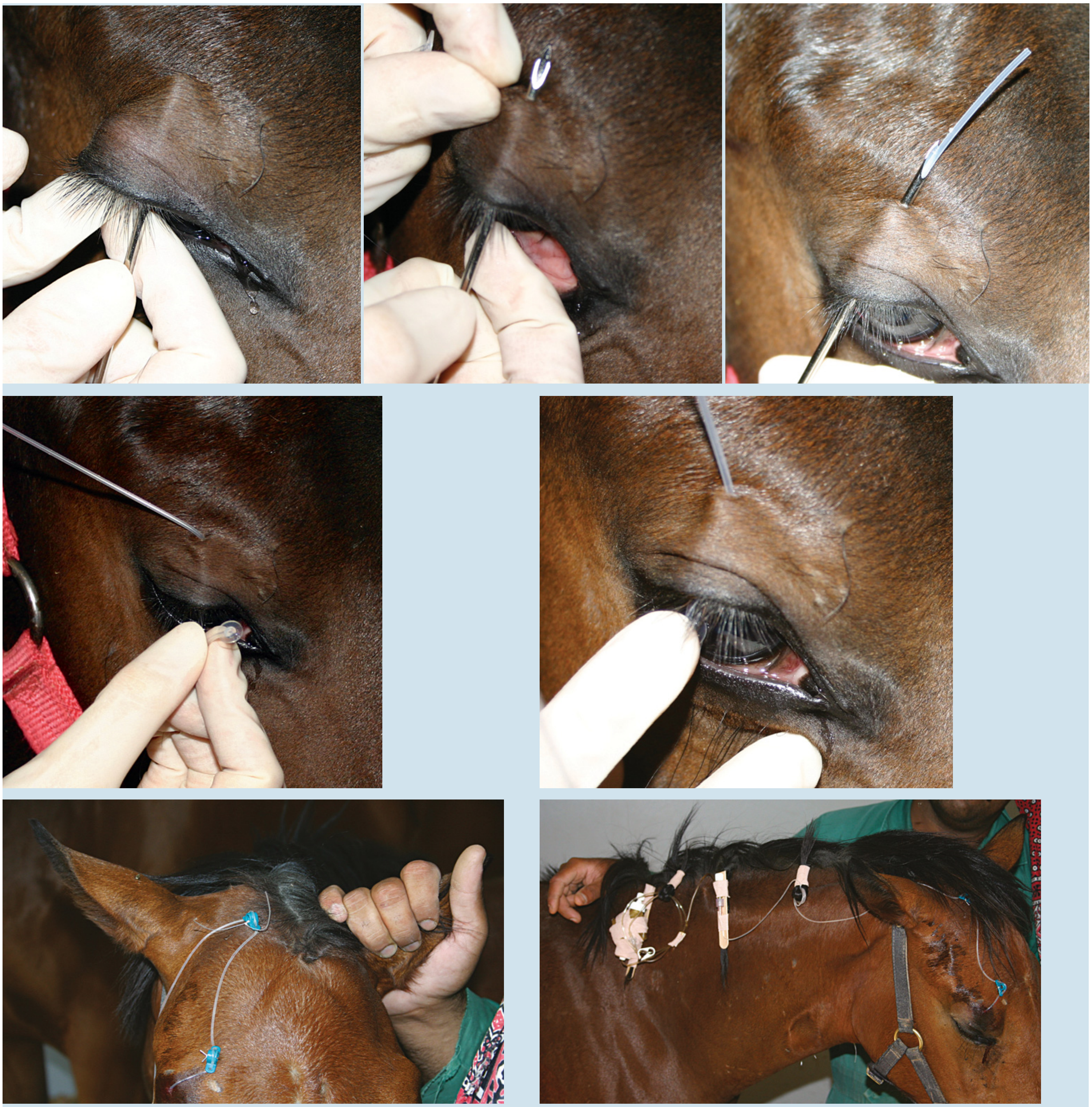
Superficial erosion involves loss or partial loss of the epithelium and usually heals in 1–2 days, being covered by a sliding or "leap frog" movement of the surrounding epithelial cell without mitosis. Superficial erosions may go undiagnosed unless stained with fluorescein because the cornea does not take on much edema and remains relatively clear

Superficial ulcer

Superficial ulcers heal rapidly by epithelial basal cell migration and subsequent mitosis. Mitosis of the newly arrived cells restores epithelial thickness, with completion of the healing process in 5–7 days.

Deep or melting ulcers

- Corneal ulcers in foals are frequently traumatic in origin and may not always be considered infected.
- Other causes may include mechanical, infectious, metabolic and neurogenic.
- The essential steps for management of corneal ulcers are to determine the cause, establish an appropriate treatment, and, if needed, protect and support the cornea.



Figures 14.34–14.40: Placement of a subpalpebral lavage system. The patient is sedated. Auriculopalpebral and frontal nerve blocks are performed and topical proparacaine (0.5 mL) is used to anesthetize the cornea and conjunctiva (see Figs 14.1–14.3).

A gloved index finger coated with ophthalmic lubricant or ophthalmic medicated ointment is placed between the globe and the upper eyelid into the fornix. A 12-gauge trocar is then run alongside of the index finger with the tip of the finger covering the proximal end of the trocar to protect the cornea. The lavage system must be properly placed within the fornix to avoid corneal abrasion. The lavage system is fed into the bottom hole of the trocar and fed through. The trocar is pulled dorsally out of the lid and the remainder of the lavage system pulled through the hole that was created by the trocar into the dorsal fornix. Once the system is placed, close the superior eyelid and watch the lavage tubing where it enters the lid. If the tubing moves a significant amount then the lavage system was not placed deep enough into the fornix.

The lavage tubing is secured just above the insertion site (superior eyelid) with tape and either suture or cyanoacrylate glue (Crazy Glue™ or Super Glue™). A 20-gauge catheter or 18-gauge luer stub adapter and PRN adapter are used at the end of the tubing as an injection port. The tube is passed through braided mane. The apparatus is then secured to a tongue depressor with tape. Drugs are administered at 0.2 mL increments and then chased with 0.8 mL of air slowly to ensure that all the medication is given.

Removal of the lavage system is done under sedation. Topical proparacaine (0.5 mL) is used to anesthetize the cornea and conjunctiva. The tape is removed from the lavage system and then the tubing is cut 3 cm above the upper eyelid. A gloved finger with ophthalmic lubricant (or medication) is then swept in the dorsal fornix to find the end plate and exteriorize it. In rare cases the footplate cannot be removed (embedded in conjunctival overgrowth) and does not appear to be a problem but could be removed by an incision in the superior eyelid.

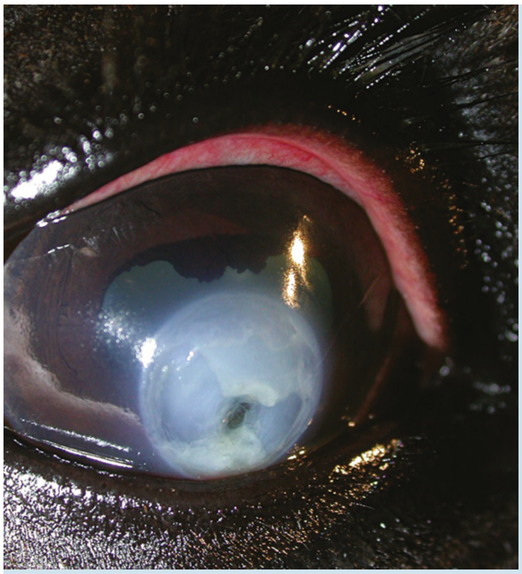


Figure 14.41: A 4-month-old Thoroughbred colt with a deep melting ulcer secondary to *Streptococcus zooepidemicus*. This image was taken after the keratomalacia was debrided with a cotton swab. Note the descemetocele in the center of the ulcer. Neomycin-bacitracin-polymyxin B and chloramphenicol ophthalmic ointment were used on a rotating basis q 4 hours. Atropine q24 hours until effect and tetanus antitoxin 0.2 mL q6 hours. The eye healed in 6 weeks. Due to financial constraints, a subpalpebral lavage was not placed in this case.

Figure 14.42: Iris prolapse in a foal that presented with a history similar to Fig 14.41. The foal maintained a visual eye with anterior synechiae in the region of the iris prolapse. Note the 9 mm superior corneal vessels. This patient had corneal disease for 12–14 days prior to presentation (corneal vessels appear 3–5 days after insult and grow approximately 1 mm a day).

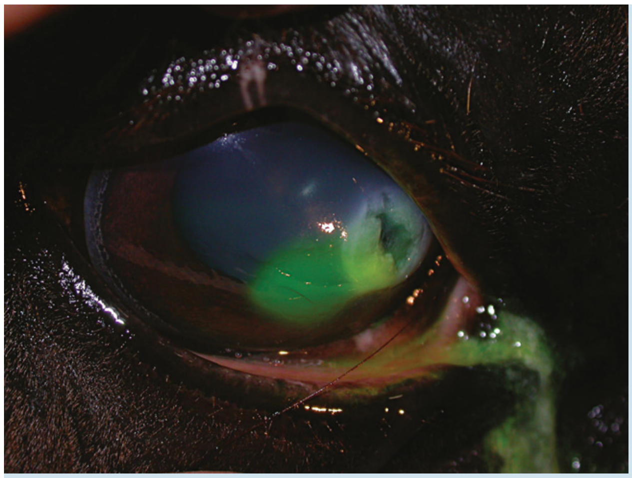


Figure 14.43: A 4-month-old colt was noted to have an acute melting ulcer of the right eye. Ulcer depth was estimated to be 85% – almost down to Descemet's membrane.

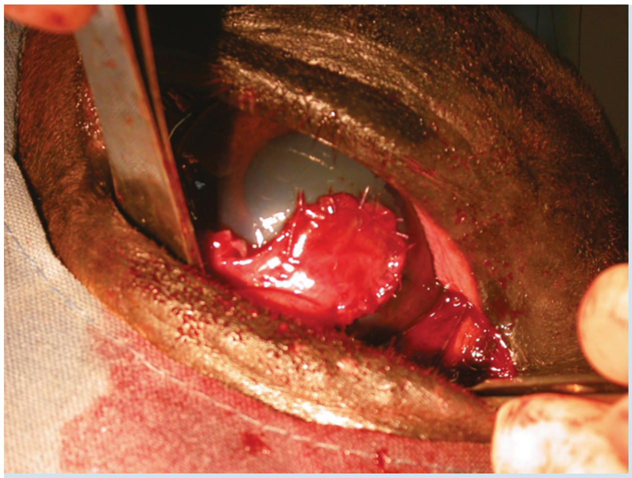


Figure 14.44: A conjunctival pedicle graft is placed over the corneal defect. Note that the graft was 2 mm larger than the actual defect (same foal as Fig 14.43). A temporary tarsorrhaphy was placed for 5 days to help minimize blinking movement and to allow the graft to adhere to the stroma.

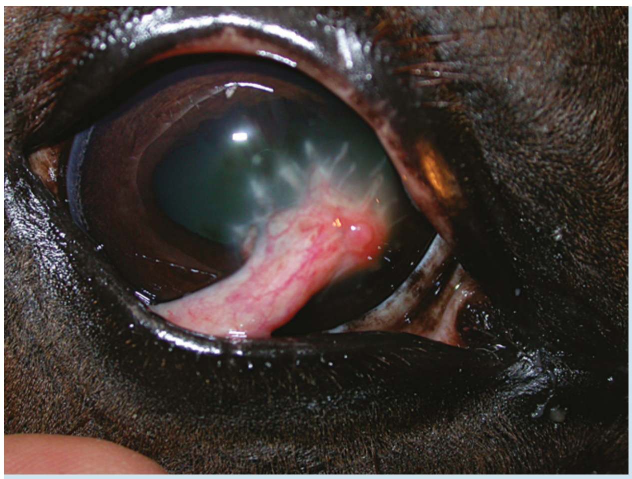


Figure 14.45: Pedicle graft 5 weeks post-surgery (same foal as Figs 14.43 & 14.44). The graft was transected at the base to interrupt its blood supply. This procedure was done with the use of sedation, topical anesthesia and Stevens tenotomy scissors. Disrupting the blood supply will allow the graft to recede and lessen the resulting corneal scar. Corticosteroids are not usually recommended, but can be used topically after the graft has been transected to minimize scar formation.

- Infectious agents may be opportunistic or invade following injury, drug therapy, or primary corneal infections. Common bacterial isolates are *Pseudomonas* spp, *Streptococcus* spp, *Staphylococcus* spp and *Bacillus*. Keratomycosis may also be encountered with agents such as *Aspergillus* spp, *Fusarium* spp, and *Penicillium* frequently isolated.

Clinical signs

These include pain, ocular discharge, and loss of corneal transparency, blepharospasm, enophthalmos, photophobia, and lacrimation.

Treatment

- The goal of treatment is to eliminate infectious and proteolytic agents, while minimizing inflammation and its sequelae and assisting in repair of the lesion.
- Treating ulcers initially with either gentamicin, chloramphenicol, neomycin or polymyxin B was found to have 90% or greater efficacy against 93 bacterial isolates in one study. Ciprofloxacin 0.3% or tobramycin 0.3% should be used in horses with severe *Pseudomonas* infections that are resistant to gentamicin.
- The use of hypertonic saline is beneficial in cases which have a large amount of corneal edema.
- Topical administration of miconazole 1%, natamycin 3.33%, fluconazole 0.2%, itraconazole 1% : 30% DMSO, amphotericin B 0.15% has been used successfully to treat fungal ulcerations.
- The frequency of administration depends on the severity of the ulcer. Superficial ulcers may have to be treated only 2–3× daily while deep and melting ulcers may have to be treated every 2 hours.
- Antiproteolytic agents commonly used include:
  - *N*-acetyl cysteine 5–10% (0.2 cc every 2–6 hours)
    - mode of action (MOA): matrix metalloproteinases inhibitor and calcium chelator
    - 5 mL 20% mucomyst in 15 mL artificial tears
  - EDTA 0.2% (0.2 cc every 2–6 hours)
    - MOA: matrix metalloproteinases inhibitor and calcium chelator
    - 5 mL of sterile water to lavender tube (EDTA)
      - If larger tube fill with sterile water until vacuum is gone
  - Serum (0.2 cc every 2–6 hours)
    - MOA: matrix metalloproteinases inhibitor and serine protease inhibitors
    - Good for 7–8 days refrigerated
    - Cheap!!
  - Tetanus *anti-toxin (NOT DILUTED: 0.2 cc every 2–6 hours)*
    - MOA: matrix metalloproteinases inhibitor and serine protease inhibitors.
- The use of a subpalpebral lavage system is very useful to aid in the medication of foals that are fractious and for foals that need medications at frequent intervals. Ulcers that have a descemetocele present will benefit from the use of a lavage system, because increased pressure on the globe when attempting to administer topical ointments may cause the cornea to rupture.
- Conjunctival grafts are needed for deep, unresponsive, melting or large corneal ulcers in addition to descemetoceles and perforated corneal ulcers.

Figure 14.46: Large stromal abscess with intense limbal vascularization.

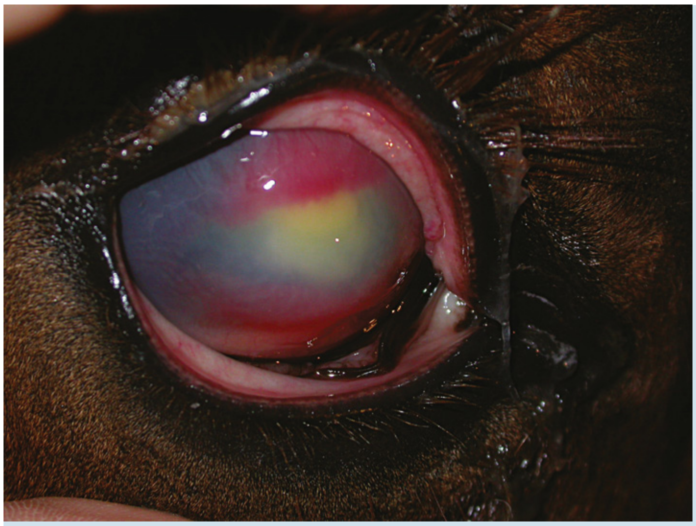


Figure 14.47: Corneal vessels penetrating (deep and superficial) a corneal abscess. Once the vessels reach the abscess it will heal properly. Systemic as well as topical antibiotics are instituted until the entire abscess is infiltrated by the corneal blood vessels.

- The melting ulcer should always be stabilized with medical therapy before surgery is pursued. This will help decrease the proteinase digestion of the absorbable sutures. The influx of PMNs, antibodies, serum and α2-macroglobulin are exuded into the corneal ulcer bed.

Corneal stromal abscess (Figs 14.46–14.52)

Corneal stromal abscesses are a chronic disorder but most affected patients usually present as an emergency. Corneal abscesses appear to develop when epithelial cells divide and migrate over focal regions of trauma or micropunctures resulting in infection of the stroma.

Figure 14.48: A 5-month-old QH filly with a stromal abscess secondary to a foreign body that penetrated the cornea dorsomedially. The foreign body was removed and a small iris prolapse occurred.

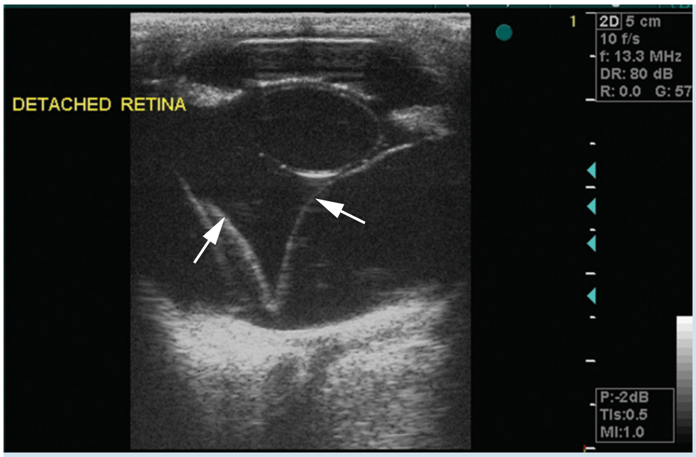


Figure 14.50: An ultrasonographic examination of the foal in Fig 14.49. Note the thickened cornea and the detached retina (arrows). This animal will not have a visual eye. The eye became phthisical after 4 months of medical therapy which consisted of a subpalpebral lavage system and topical antibiotics (Neopolybac® and chloramphenicol).

Figure 14.49: Severe abscessation of the entire cornea. A central ulcer is present down to Descemet's membrane. This abscess and ulcer healed with a large scar.

Inappropriate use of topical corticosteroid therapy may also predispose the cornea to stromal abscess formation.

Clinical signs

- A corneal stromal abscess appears as a focal, yellow to white, stromal infiltrate usually with corneal edema.
- Mild to marked blepharospasm secondary to iridocyclitis with epiphora is usually noted. Fluorescein dye retention may or may not be seen.
- Corneal vascularization is variable at presentation.
- Hypopyon and fibrin accumulation in the anterior chamber is not uncommon.

Diagnosis

- Diagnosis is primarily based on clinical signs.
- It may be difficult to determine an etiological agent with a corneal scraping and cultures unless the abscess is superficial and it is not recommended to perform deep corneal debridement.

Treatment

- Stromal abscesses do not heal completely until they are infiltrated by corneal blood vessels.
- Treatment consists of antimicrobial and antifungal therapy. Routine antifungal drugs do not reach sufficient corneal concentrations unless they are compounded with DMSO (1% Itraconazole : 30% DMSO Topical Ophthalmic Ointment). However this combination may cause some irritation when applied topically. Fluconazole (6 mg/kg PO q24 h) has also been used. This is given for a minimum of 1 week but in many instances may be required for much longer.
- Lipophilic ophthalmic antibiotics such as chloramphenicol, ciprofloxacin, and polymyxin/oxytetracycline may be able to achieve higher corneal concentration when compared to other antibiotics. The author tends to combine chloramphenicol and neomycin/polymyxin B/bacitracin as topical antimicrobials with good success.
- Debridement of the corneal epithelium may increase drug concentration in the cornea and aqueous humor.
- Systemic broad-spectrum antibiotics may also be of benefit when corneal vessels are present, allowing an increased antimicrobial concentration of the cornea's stroma. Atropine 1% (to effect every 4–6 hours) combined with systemic anti-inflammatories such as phenylbutazone or flunixin meglumine would be needed in cases with iridocyclitis.
- In severe cases, a penetrating keratoplasty or posterior lamellar keratoplasty may be warranted.
- Treatment is generally required for 6–8 weeks with healing resulting in significant scar formation.

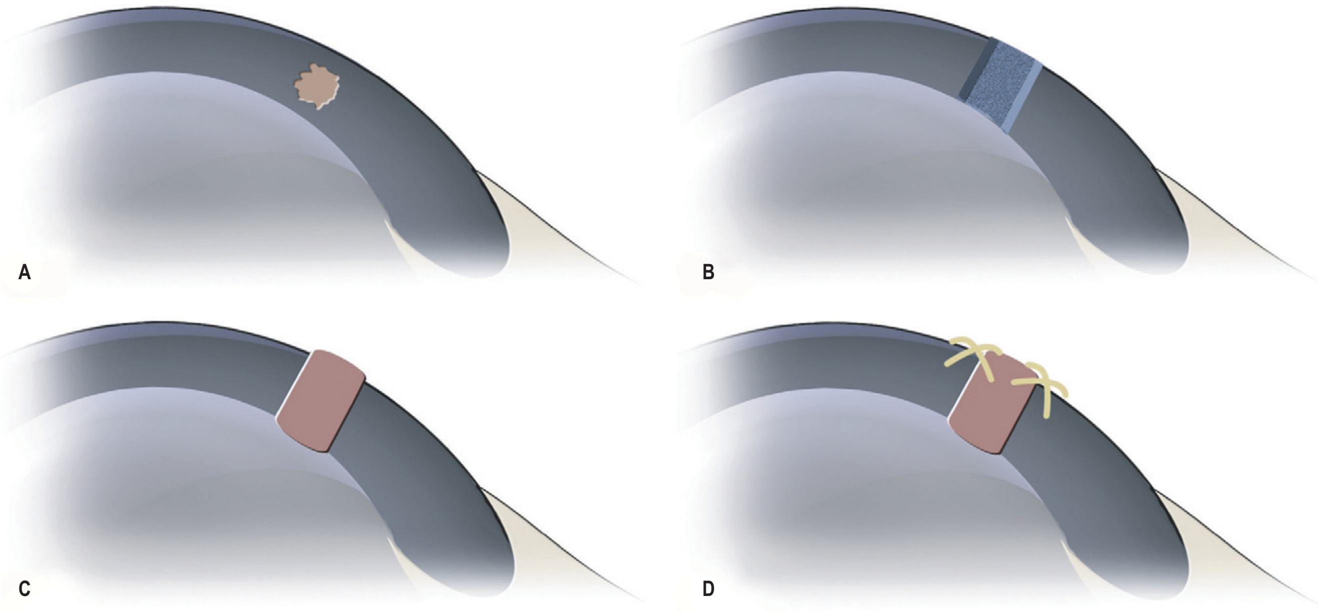


Figure 14.51: Penetrating keratoplasty. (a) Stromal abscess involving most of the corneal thickness. (b) Removal of the entire corneal thickness in PK procedure. (c) Placement of full thickness corneal graft. (d) Suture placement in the corneal graft. (From Brooks DE 2005 Clinical Techniques in Equine Practice: Equine Ophthalmology Volume 4, Number 1, p 39.)

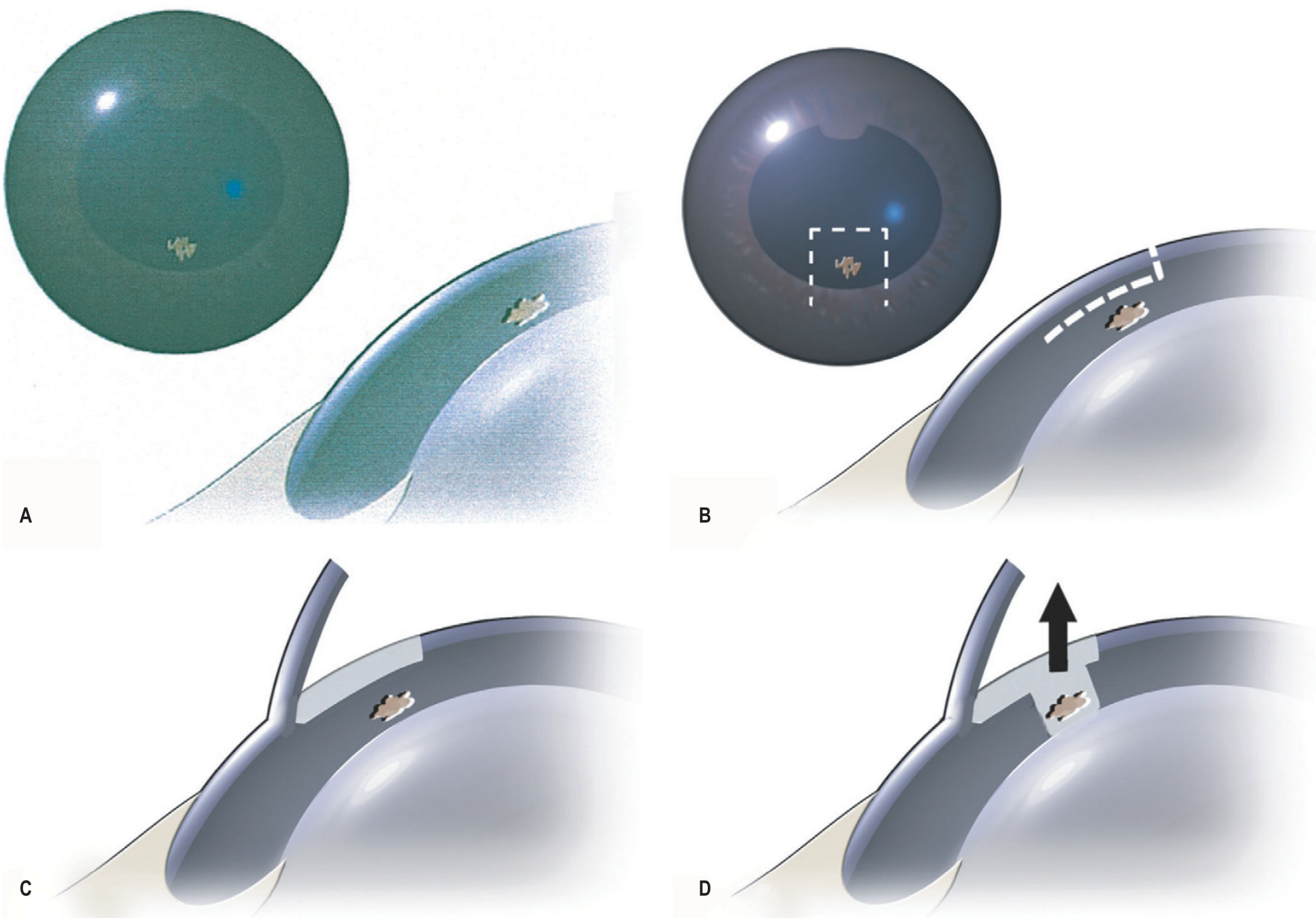


Figure 14.52: Posterior lamellar keratoplasty. (a) Stromal abscess in axial cornea. (b) Superficial incisions in PLK procedure. (c) Abscess exposure in PLK. (d) Abscess removal of PLK. (From Brooks DE 2005 Clinical Techniques in Equine Practice: Equine Ophthalmology Volume 4, Number 1, p 41.)

Figures 14.53 & 14.54: Foal with panuveitis secondary to *Salmonella* enterocolitis. Note the green hue of the iris and fibrin in the anterior chamber. Topical steroids and atropine helped resolve the panuveitis.

Uveitis (Figs 14.53 & 14.54)

Uveitis in the foal occurs secondary to a variety of systemic conditions. Inflammation of the uveal tract of the eye can occur in a foal suffering from trauma to the globe, systemic endotoxemia, colitis, rhodococcus pneumonia, corneal laceration/ulceration, leptospirosis and in other causes of systemic inflammation.

Clinical signs

- Miosis
- Scleral injection
- Blepharospasm
- Photophobia
- Corneal edema
- Keratic precipitates.

Treatment

Treatment has three main aims:

- Reducing discomfort, which can be achieved with mydriatic cycloplegics such as topical atropine.
- Decreasing inflammation of the uveal tract through the use of systemic anti-inflammatories such as flunixin meglumine or phenylbutazone and/or topical corticosteroids or anti-inflammatories.
- Determining and treating the underlying cause.

Glaucoma (Figs 14.55–14.57)

Glaucoma is a disease that results in increased intraocular pressure (IOP) that if severe enough will cause irreparable visual deficits of the eye. Glaucoma can be congenital, primary or secondary.

Congenital equine glaucoma has been reported in Thoroughbred, Arabian and Standardbred foals. Secondary glaucoma in foals is most commonly associated with trauma and intraocular inflammation (uveitis).

Typically glaucomas are slow in progression with little evidence of pain and insidious loss of vision. Acute cases of glaucoma are usually related to intraocular trauma.

Normal IOP in horses is 20–25 mmHg. Failure to utilize auriculopalpebral nerve blocks during tonometry in horses may result in slight overestimation of IOP. Horses sedated with xylazine have been shown to have IOP decreased by 23–27%.

Clinical signs

- Clinical signs may include corneal edema, buphthalmos and epiphora.
- Fixed dilated pupil or a miotic pupil.
- Congenital glaucoma may be bilateral.
- Lens luxation, lens coloboma, absent corpora nigra, iris hypoplasia, retinal degeneration and optic nerve cupping may be present.

Treatment

- Treatment for congenital glaucoma is usually unrewarding because of the high incidence of ocular congenital abnormalities associated with this disease.
- The goal of therapy is to reduce the IOP. Atropine can be used to aid in reducing IOP. If severe the use of a beta-blocker (timolol maleate 0.5% q12–q8 hours) and/or carbonic anhydrase inhibitor (dorzolamide 2.0% q 12–q8 hours) may be used.
- Iridocyclitis must also be treated if present. The use of anti-inflammatories consisting of topical corticosteroids such as prednisolone acetate or dexamethasone and systemic anti-inflammatories such as phenylbutazone and flunixin meglumine is

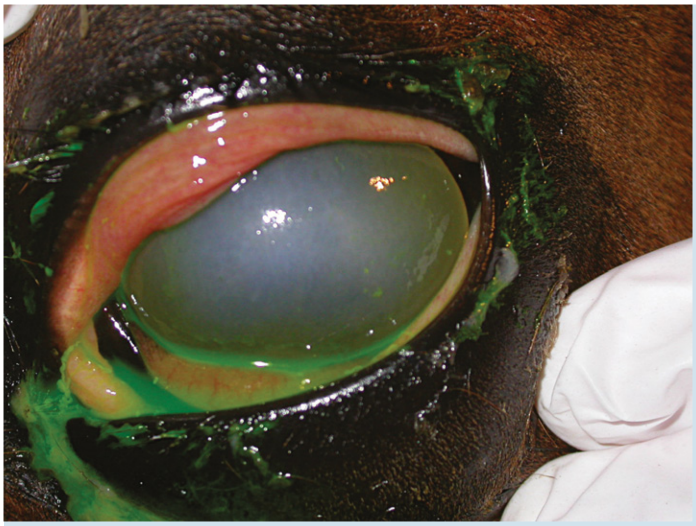


Figure 14.55: Glaucoma with corneal edema and mild chemosis. No corneal ulceration was noted. Intraocular pressure (IOP) of the left eye was 35 mmHg, IOP of the right eye was 23 mmHg.

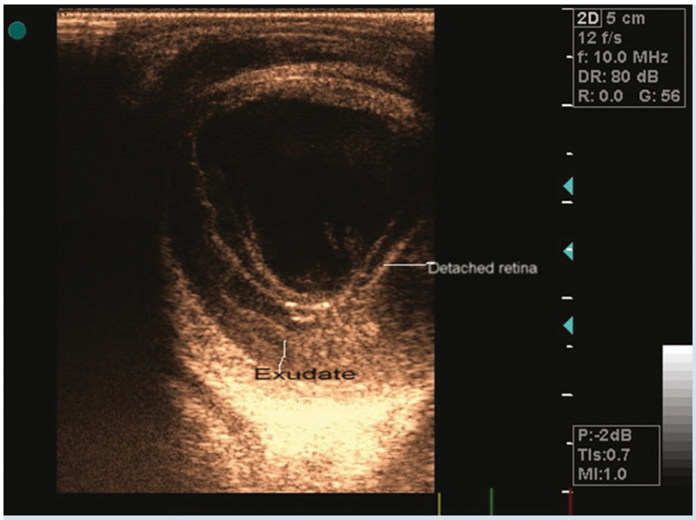


Figure 14.57: Ocular ultrasonography of the left eye of the foal in Fig 14.56 revealed marked panuveitis with a detached retina and exudate posterior to the retina. The foal was started on systemic antibiotics (potassium penicillin and gentamicin) and flunixin meglumine. Atropine and hypertonic saline were applied topically. The foal's eye became phthisical 4 weeks after the initiation of therapy.

Figure 14.56: Buphthalmos, blepharoedema and epiphora of the left eye in a 4-month-old TB colt that was found with a painful eye in the pasture. The glaucoma in this case was considered to be secondary to trauma.

effective in the control of iridocyclitis causing glaucoma. A penetrating keratoplasty or posterior keratoplasty may be implemented on corneal abscesses that do not respond to conventional therapy.

Eyelid lacerations (Figs 14.58–14.60)

Eyelid lacerations will be encountered by every equine practitioner. Upper eyelid damage is more significant because it covers more surface area of the cornea than the lower eyelid.

Clinical signs

Clinical signs associated with an eye lid laceration are obvious. An ocular exam will be necessary to rule out other injury such as punctured globe, corneal ulcers or uveitis.

Treatment

- An eyelid laceration is a surgical entity.
- Partial thickness lacerations can be closed with a single layer of 4-0 to 6-0 non-absorbable, monofilament suture.
- Full thickness lacerations involving the eyelid margin require meticulous two-layer closure. Minimal tissue debridement should be performed, especially of the eyelid margin proper. Excision of a portion of the lid or lid fragment should be avoided because the tremendous blood supply to the lid margin can be vital in healing hopeless appearing injuries. Primary repair should occur within 24 hours for optimal functional and cosmetic results. Hypertonic saline ophthalmic ointment can be placed on the exposed soft tissue to reduce the amount of chemosis and/or edema present. It is recommended that the skin closure is started at the eyelid margin to allow perfect apposition without a "step." A figure-eight suture

Figure 14.58: Full thickness laceration of the inferior eyelid. No corneal ulceration was noted. Eyelid laceration was aseptically prepared for sutures.

Figure 14.59: Eyelid after repair.

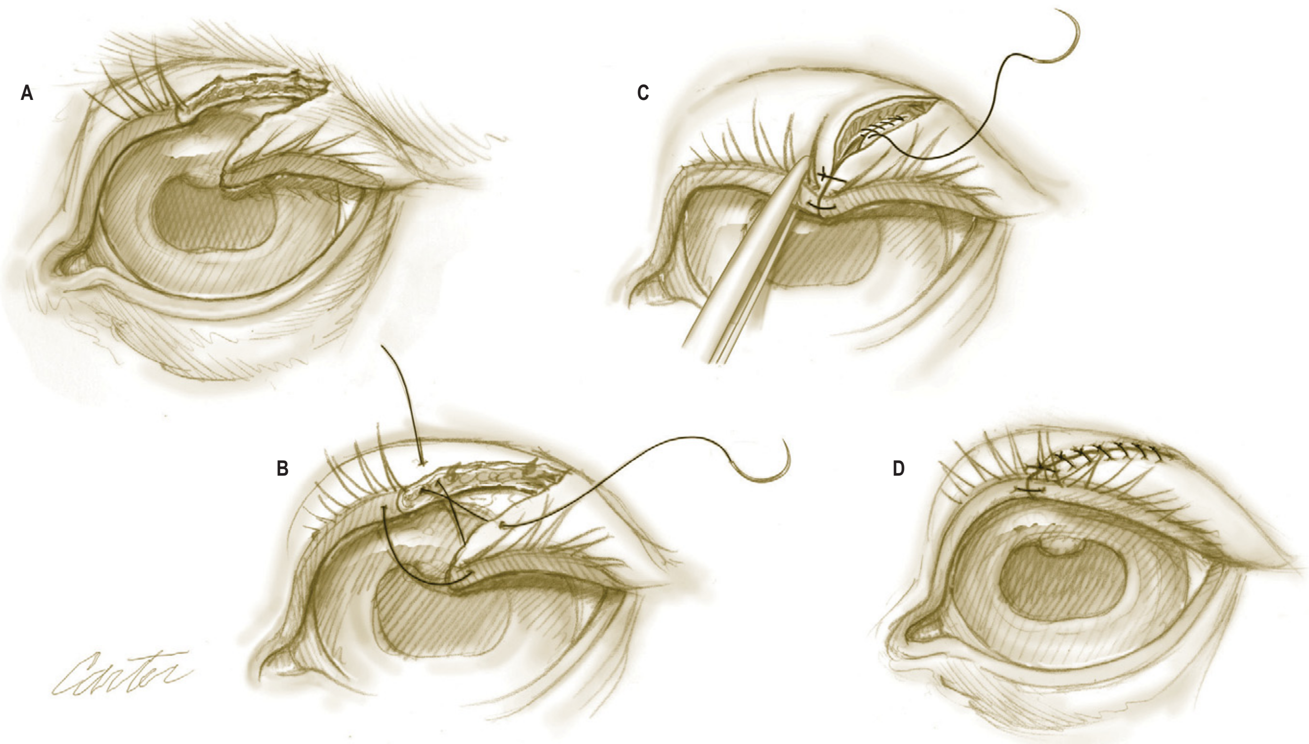


Figure 14.60: Eyelid laceration repair. (a) The wound is cleansed and minimally debrided. (b) A figure-eight suture at the eyelid margin will allow for alignment of the eyelid margin, which is critical to an effective repair. (c) A two-layer closure is performed if possible. The deep layer should start at the eyelid margin as well and proceed distally from it. (d) Simple interrupted skin sutures finish the closure. (From Capaldo F, Komáromy AM 2006 Clinical Techniques in Equine Practice Volume 5, Number 2, p 135.)

pattern or a mattress pattern can be used to appose the eyelid margins. The conjunctiva is closed initially with absorbable 5-0 to 6-0 suture in a continuous pattern. The skin should be closed with 5-0 to 6-0 non-absorbable monofilament suture. A temporary tarsorrhaphy may be used to limit exposure of the globe if eyelid swelling is severe.

- Medical therapies consist of tetanus prophylaxis, systemic antibiotics, systemic anti-inflammatory therapy for 3–5 days.
- Topical broad-spectrum antibiotics are only necessary if the cornea is involved.
- Prognosis for eyelid lacerations when treated appropriately is excellent.

Recommended reading

Brooks D 2005 Equine ophthalmology. Clinical Techniques in Equine Practice 4(1):2–11, 21–29, 50–72

Cutler T 2004 Updates in ophthalmology. Veterinary Clinics of North America, Equine Practice 20(2):285–297

Gilger B 2005 Equine ophthalmology, 2nd edn. WB Saunders, Philadelphia

CHAPTER 15

The nervous system

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Introduction

Abnormal behavior patterns, seizures and weakness are commonly encountered in neonatal foals. Such clinical signs may be referable to the nervous system or may be secondary to disorders of other body systems. Definitive diagnosis of a neurological disorder may be challenging and in many foals there may be both a primary and secondary disorder, e.g. a foal suffering from both hypoxic–ischemic encephalopathy and hypoglycemia. Similarly the onset of neurological signs in older foals can present a diagnostic challenge. Maternal antibodies have begun to wane making the foal more susceptible to many infectious agents, but they can still be affected by many of the disorders that affect younger foals.

A thorough clinical examination including complete blood count, serum biochemistry and electrolytes is essential in any foal demonstrating abnormal behavior patterns. Repeat physical examinations (and blood analysis where required) should be at short time intervals as foals can show rapid deterioration in clinical condition within a matter of hours. The objectives of the initial examination are to determine:

- Is a primary neurological disorder present? This involves not only a thorough neurological examination but also examination of other body systems and may involve the use of adjunct examinations.
- What is the location of the neurologic lesion? Single or multifocal?
- What is the nature of the disease process involved? Is it infectious or non-infectious? Traumatic? Metabolic? Other?
- Formulation of a treatment plan.

Subsequent evaluations should assess whether there has been an improvement in the clinical condition of the foal and should assess the treatment protocol; e.g. if seizures are present are they being controlled by the medication that is being given?

Neurological examination

See Chapter 3 (pp. 56–58).

Adjunct examinations

Cerebrospinal fluid analysis (Figs 15.1–15.4)

CSF can be collected from the atlanto-occipital space using a 1.5-inch, 20 g disposable hypodermic needle with the needle placed midline, just cranial to the cranial borders of C2. The subarachnoid space is small and the needle is placed in close proximity to the brain stem – supervised practice is strongly advised.

CSF should be clear, can be slightly xanthochromic (yellow coloration) in foals, have less than 6 mononuclear cells per µL and a protein content of 50–100 mg/dL. Infection can result in CSF pleocytosis and elevations of protein content and decreased glucose content.

Trauma and vascular disease can result in hemorrhage, leading to a yellow coloration (xanthochromia) which persists even when red

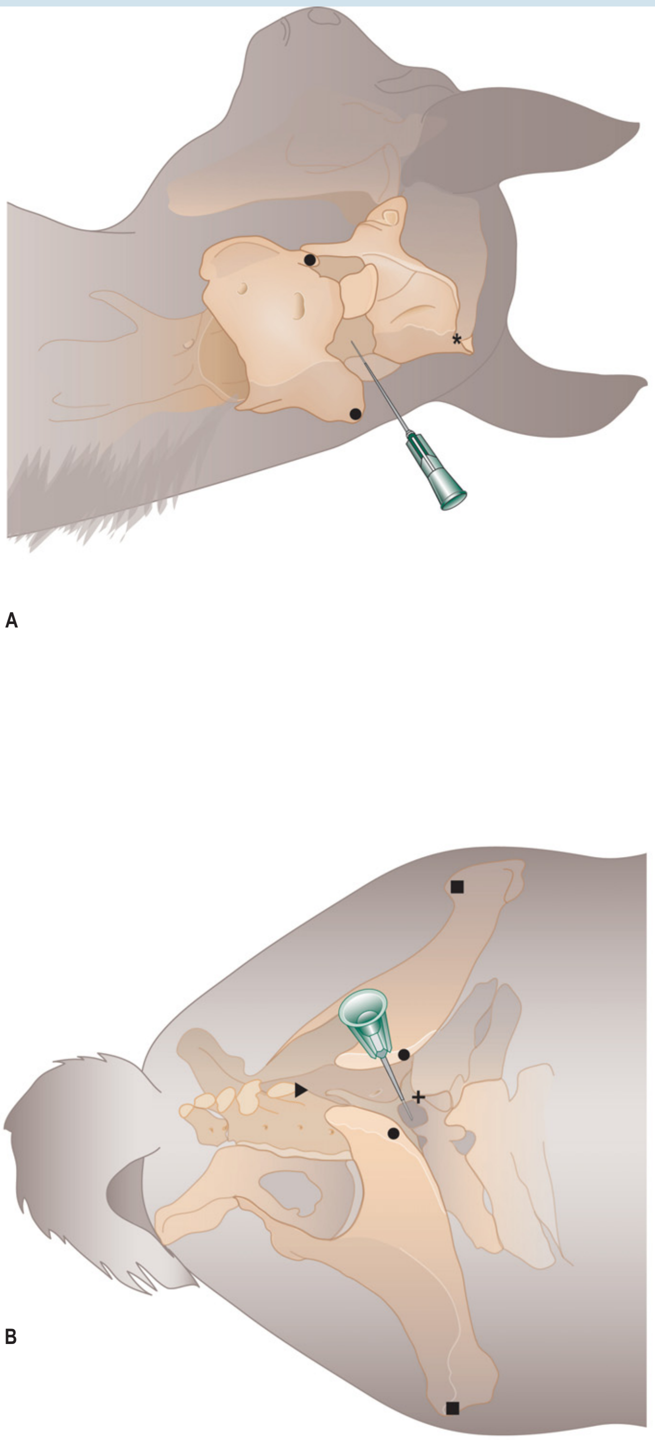

Figure 15.1: (a) Atlanto-occipital collection of CSF fluid. The head is flexed towards the chest and two imaginary lines are drawn. One line joins the cranial edges of the wings of the atlas (dots) and the second line is drawn along the midline from the occipital protuberance (*) to bisect the first line. The needle is inserted just cranial to the bisection of the two lines and directed towards the lower lip. (b) Lumbosacral collection of CSF fluid. Foals are normally sedated and placed in lateral recumbency with the hindlegs pulled slightly forward. The site is a palpable depression on the midline immediately behind L6, just cranial to a line drawn between the palpable cranial edges of each tuber sacrale (dots). Other palpable landmarks are the caudal borders of each tuber coxae (squares) the caudal edge of the spine of L6 (cross) and the cranial edge of the second sacral spine (triangle). After aseptic preparation and infiltration with local anesthetic the spinal needle is introduced perpendicularly to a depth of 3–4 cm in neonates. A loss of resistance and twitching of the hindlimbs indicates entry into the subdural space.

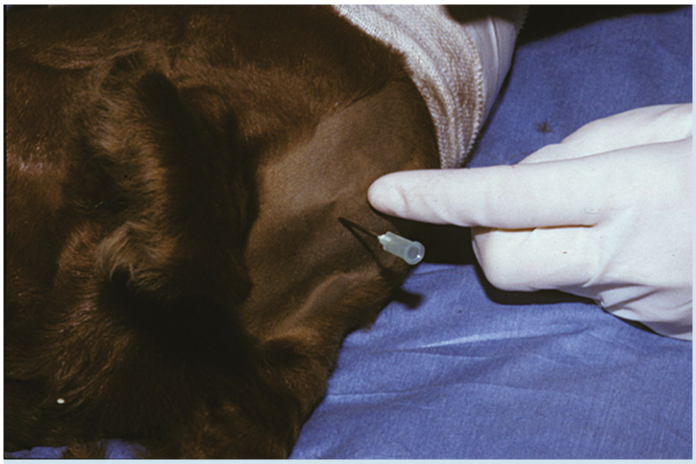

Figure 15.2: Atlanto-occipital spinal tap in a foal.

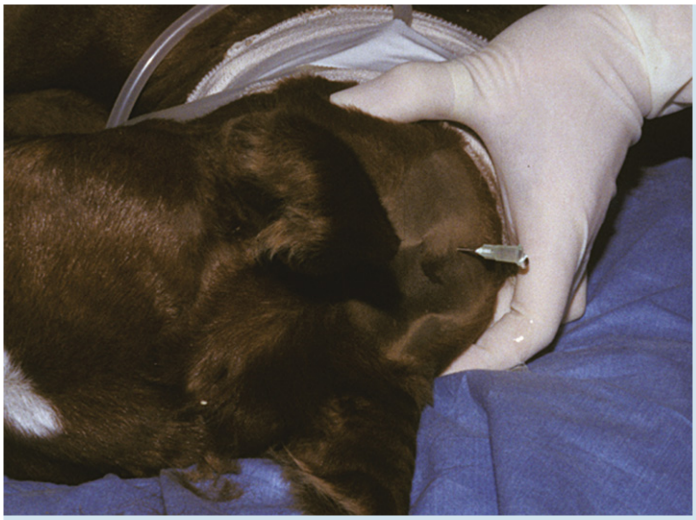

Figure 15.3: Same foal as in Fig 15.2; note the clear spinal fluid.

Figure 15.4: Atlanto-occipital spinal tap in an adult horse post trauma. Note the large quantity of blood in the spinal fluid.

cells have been spun off, as well as infiltration by non-toxic neutrophils and macrophages.

Neuroradiology (Figs 15.5–15.7)

Plain radiography of the vertebral column and skull is indispensable for identifying bony malformations including cervical vertebral malformation, fractures and osteomyelitis. Myelography is rarely indicated and should only be attempted in referral hospitals – and then only if the treatment protocol will be substantively changed depending on the result.

Ultrasound examination of the atlanto-occipital space (Figs 15.8–15.11)

By Stefania Bucca (DVM), Ursula Fogarty (MVB, PhD, MRCVS, DIPStat) and Brendan T. Farelly (MSc, DMedVet, MRCVS)

Although CNS clinical signs are a common clinical manifestation in the neonatal foal, it is currently difficult to routinely evaluate the CNS without using sophisticated or invasive procedures. During a study to evaluate CSF pressure in neonatal foals it was observed that ultrasound examination at the atlanto-occipital space provided useful information on the CSF and associated structures. It was possible to assess CSF quality and ultrasound measurements taken at this location showed a correlation with CSF pressure, measured using a spinal monometer.

The ultrasound technique was developed and evaluated at the Irish Equine Centre, the Hagyard Equine Medical Institute in Kentucky, and at a veterinary practice in Italy. Linear or microconvex, real-time ultrasound technology mounted with a 7.5–8 MHz transducer, was used to assess the atlanto-occipital space. Standing and recumbent foals were restrained with their head flexed and alcohol was liberally sprayed or sponged on behind the poll. Clipping was usually not necessary. Cross-sectional and longitudinal views of the atlanto-occipital space were obtained by holding the transducer perpendicular to the area.

Structures identifiable at this site included the dorsal cervical musculature, occipital condyles, and subcutaneous tissue, the spinal

Figures 15.5 & 15.6: Positioning for obtaining neutral survey radiographs of the cervical area. If measurements are to be taken such as in CVM cases it is important to obtain true laterals which requires extension of the neck (Fig 15.6).

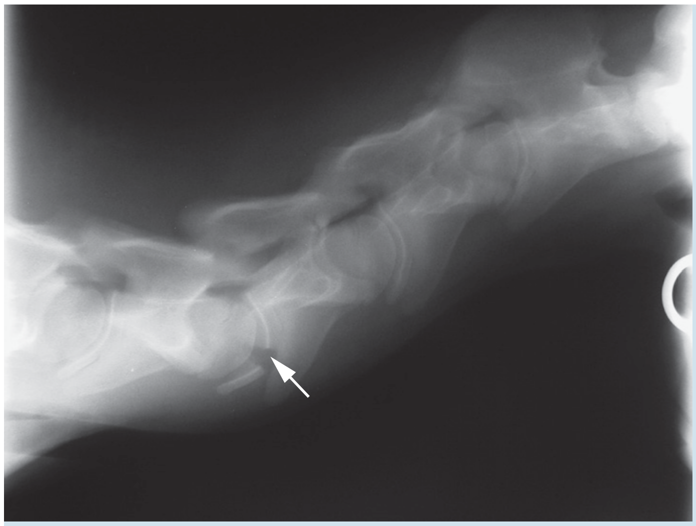

Figure 15.7: Plain radiography of the cervical spine in a foal demonstrating a fracture of C4 vertebrae (arrow).

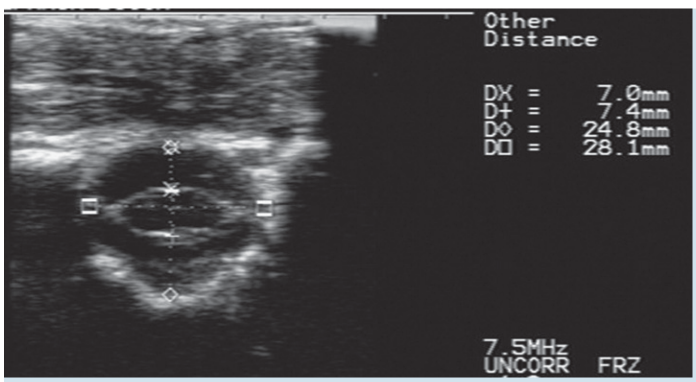

Figure 15.8: Ultrasound cross-sectional scan of a normal foal at the atlanto-occipital space. ◊ → ◊ and □ → □ denote the height and width (respectively) of the vertebral canal. □ → □ transects the spinal cord and X → X delineates the depth of the dorsal subarachnoid space at this level in the cisterna magna.

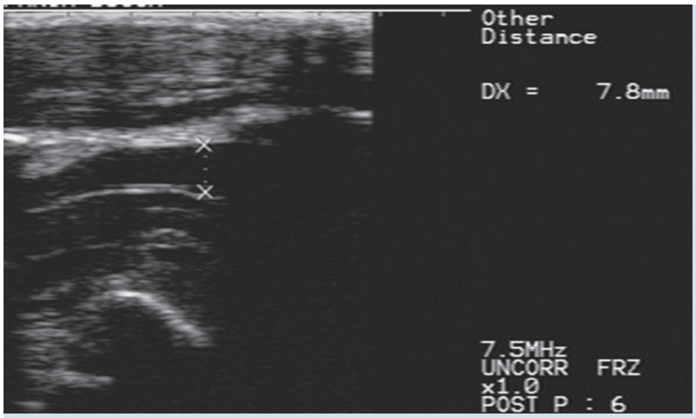

Figure 15.9: Ultrasound longitudinal scan of a normal foal at the atlanto-occipital space. X → X delineates the depth of the dorsal subarachnoid space at this level in the cisterna magna.

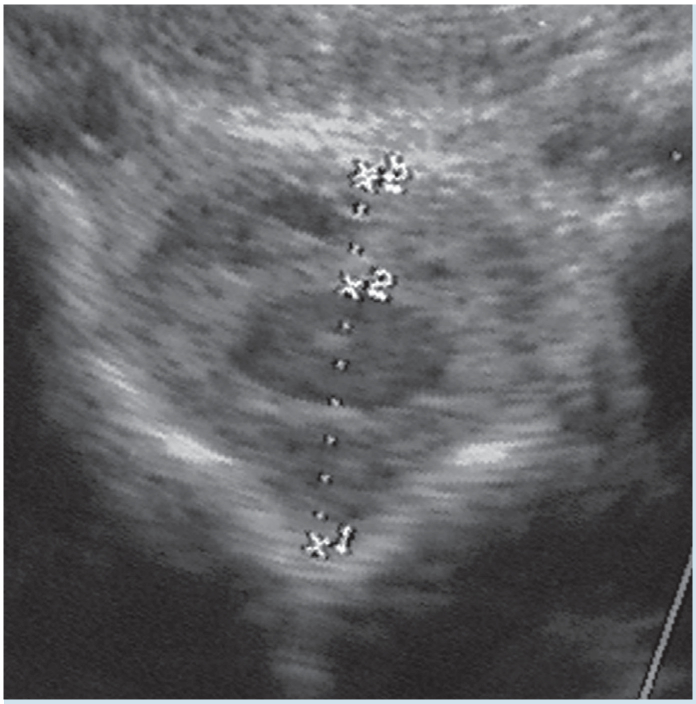

Figure 15.10: Ultrasound cross-section of the atlanto-occipital space in a foal with septic meningitis showing a marked increase in the cellularity of the CSF. A subsequent CSF tap and CSF analysis revealed a marked elevation in white cell count.

cord, meninges, the postero-ventral opening of the occipital bone and the longitudinal vertebral sinuses.

Measurements of CSF pressure taken using a spinal monometer showed a 0.77 correlation with ultrasound measurements of the dorsal subarachnoid space at this level. Ultrasound measurements to date indicate that a reading of 6.73 ± 0.51 mm may be typical of normal neonatal foals while measurements of 8.65 ± 0.86 mm were recorded in clinically abnormal neonatal foals. The quality of CSF and related structures can also be assessed by ultrasound at this site with echogenic particles suggesting cellular infiltration. Information generated using this technique can help differentiate meningitis, hemorrhage and increased cerebral spinal fluid pressure in foals presenting with neurological signs.

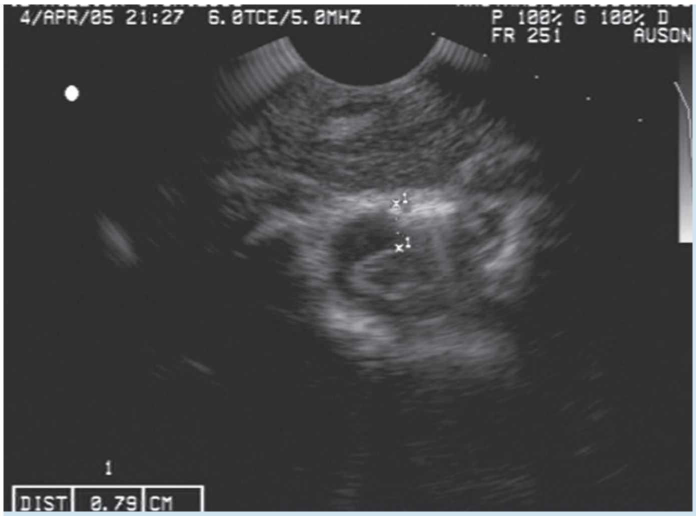

Figure 15.11: Ultrasound cross-sectional scan of the atlanto-occipital space in a foal with a focal meningeal hemorrhage or area of edema seen as hyperechoic area in the dorsal subarachnoid space.

Brainstem auditory evoked responses (Figs 15.12 & 15.13)

The brainstem auditory evoked response (BAER) test measures responses in brain waves that are stimulated by clicks in the ear to check the auditory pathways of the brainstem.

Computer averaging over time to filter background noise generates an averaged response of the auditory pathway to an auditory stimulus. Several waves are plotted for each ear and represent specific anatomical points along the auditory neural pathway – the cochlear nerve and nuclei (waves I and II), trapezoid nuclei (wave III), lateral lemniscus (wave IV), and caudal colliculi (wave V).

Delays of one side relative to the other suggest a lesion in the 8th cranial nerve between the ear and brainstem or the brainstem itself.

Normal values have not been established for foals, however marked delays can be predicted from normal adult horse values. The technique is available in specialist centers and could be useful to determine deafness in overo colored horses with congenital deafness or acquired diseases such as EPM.

Electromyography

Needle electromyography (EMG) involves placing needle electrodes in the cranial, paravertebral, and limb muscles to detect muscle fibers displaying abnormal electrical activity after disruption of their nerve supply. (See Chapter 8, p. 213.)

It does not provide a definitive diagnosis of the disease process present but can provide useful information for selecting further diagnostic tests, such as the most suitable site for nerve or muscle biopsy.

It can usually be carried out on a conscious animal with the use of some restraint such as placing in a stocks or administering a sedative agent. Commonly used sedatives such as xylazine and acepromazine do not interfere with EMG results.

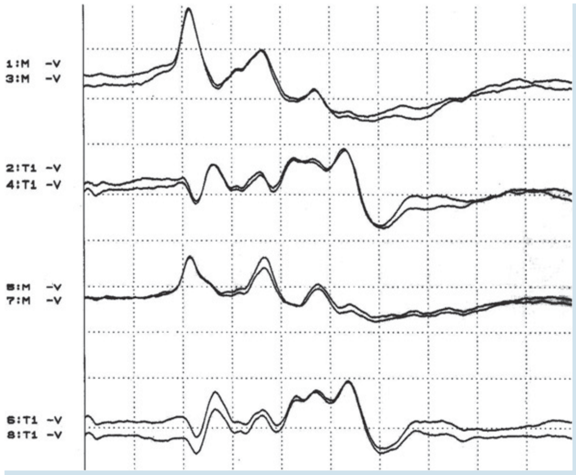

Figure 15.12: BAER (brainstem auditory evoked response) from a normal foal.

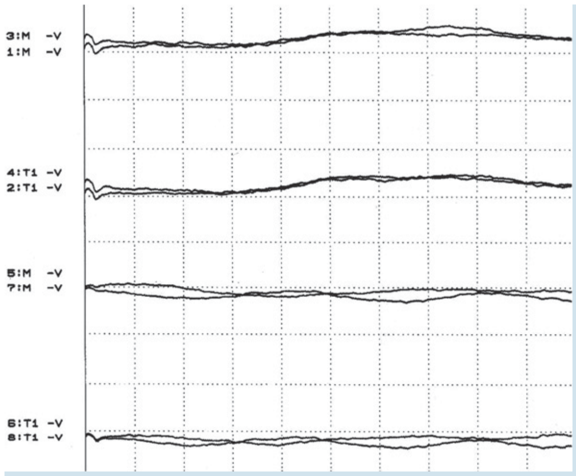

Figure 15.13: BAER from a deaf lethal white foal. Note the flat lines indicating no response to testing.

Computed tomography (Figs 15.14 & 15.15)

Computed tomography involves the creation of cross-sectional radiographic images within a circular gantry by the generation of x-rays from a rotating x-ray tube.

A series of two-dimensional transverse images are created. These images can be reconstructed in other planes and can also be used to generate three-dimensional images that may then be used to assess lesions or density patterns and can be used for surgical planning.

A number of different types of scanners are available, all of which currently require the horse to be anaesthetized and are generally only available at referral institutions.

The entire body can be scanned in foals weighing less than 200 kg. It is most commonly used for lesions of the head, neck and distal extremities.

Two of the greatest advantages of CT are that it has the ability to image distinct structures in the head, and spinal lesions that may be difficult to diagnose by other modalities can often be clearly seen with CT.

Figure 15.14: Computed tomography being performed on the head and neck of a 3-month-old foal that was thought to have cervical fracture that was not evident on plain radiographs. A fracture at C6–C7 was confirmed on the basis of the CT images.

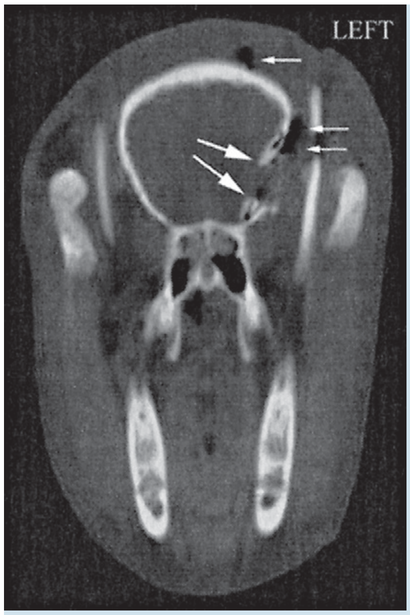

Figure 15.15: Transverse CT image at the level of the cranial aspect of the calvarium, in a 4-month-old Appaloosa filly that presented 3 days following head trauma. Survey radiography failed to identify any fractures of the calvarium. Note the compressed fractures of the left calvarium within the squamous temporal bone (large arrows). There is also emphysema within the soft tissues surrounding the fracture site (small arrows). Based on the severity of CT findings and grave prognosis, owners decided upon euthanasia. Necropsy confirmed the CT findings in addition to a secondary encephalitis. (From: Tucker RL, Farrrell E 2001 CT and MRI imaging of the equine head. Veterinary Clinics of North America: Equine Practice 17(1):133.)

Magnetic resonance imaging

Magnetic resonance imaging uses the magnetic properties of tissues to generate images. MRI images have outstanding tissue contrast and as such can provide diagnostic advantages over other imaging modalities. To date MRI has been used mainly for orthopedic imaging but the spectrum of applications is increasing as both operators and horse owners are becoming more familiar with the technique.

However, MRI is only available at a few institutions worldwide, may be cost prohibitive in many cases and anaesthesia is necessary to obtain the images.

Inherited / congenital conditions

Myelodysplasias

History

Myelodysplasias are developmental anomalies of the spinal cord that may result in clinical neurologic disease. Gross malformations of the axial skeleton tend to accompany severe anomalies of the spinal cord. An example that is occasionally seen in horses is spina bifida which results from failure of fusion of the two halves of the dorsal arch, which includes the spinous processes. This may or may not be accompanied by protrusion through the vertebral defect of cystic dilations of the spinal cord (myelocele), meninges (meningocele) or both (meningomyelocele).

Other forms of myelodysplasia may occur with or without vertebral anomalies. These may include varying degrees of syringomyelia (tubular cavitations), hydromyelia (dilation of the central canal), or diplomyelia (spinal cord duplication). Similarly vertebral malformations and vertebral column defects may occur without myelodysplasia and are not normally associated with neurologic deficits.

Clinical signs

- Myelodysplasias may be clinically evident at birth or manifested soon after as stable neurologic abnormalities such as paraparesis.
- A bunny-hopping gait and bilaterally active reflexes in the limbs at the level of the defect are prominent features.
- Progressive neurologic defects resulting from spinal cord compression is associated with severe vertebral anomalies.

Encephalocele / meningocele (Fig 15.16)

History

Prolapse of the meninges with parts of the brain (encephalocele) occurs when the overlying cranium and skin fail to close during development.

Clinical signs

- The defect may be small or may involve most of the cranium and is generally on the midline.
- The abnormality may occur alone or accompany other developmental defects.
- Affected foals are usually either aborted, born dead or die soon after birth.

Treatment

Affected foals that are born alive should be euthanized as the condition is incompatible with life.

Occipitoatlantoaxial malformation (OAAM) (Fig 15.17)

History

OAAM is reported in Arabians, Morgans, Standardbreds and rarely in other breeds and includes fusion of the atlanto-occipital joint, atlantalization of the axis and hypoplasia of atlantal wings. Foals can

Figure 15.16: Encephalocele in a newborn foal.

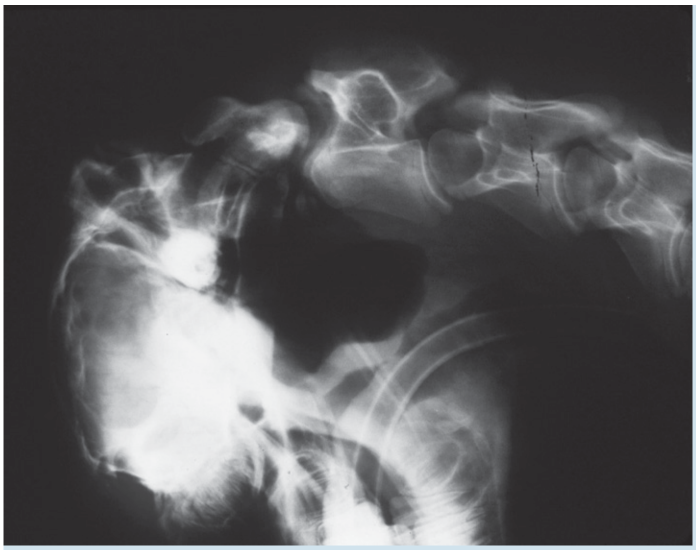

Figure 15.17: Atlanto-occipital malformation (fusion of the atlanto-occipital joint) in an Arabian filly.

be born dead, be ataxic at birth or show progressive ataxia as yearling animals.

Clinical signs

- Animals often have extended neck posture with reduced flexion of the atlanto-occipital junction.
- A malformation of C1 and C2 can be palpated.
- Usually there are varying degrees of cervical spinal cord signs (tetraparesis and ataxia in all four limbs).
- Occasionally restricted neck movements form the only clinical sign.

Differential diagnoses

Cervical fractures and cervical vertebral malformation should be ruled out.

Diagnosis

The disease can be confirmed by plain radiography. Malformations of the occiput, C1 and C2 are present including atlanto-occipital fusion and hypoplasia of the dens. Healed atlantoaxial fractures can mimic OAAM.

Treatment

No treatment is indicated and since the disease is most likely inherited in the Arabian, the owners should be counseled not to breed close family lines.

Cerebellar hypoplasia (Fig 15.18)

History

- Cerebellar syndromes and degenerative lesions are seen in Thoroughbred and Paso fino foals.
- Unlike ruminants no toxin or virus has been linked with abnormalities of cerebellar development in foals and congenital malformation or hypoplasia of the cerebellum appears to be rare.

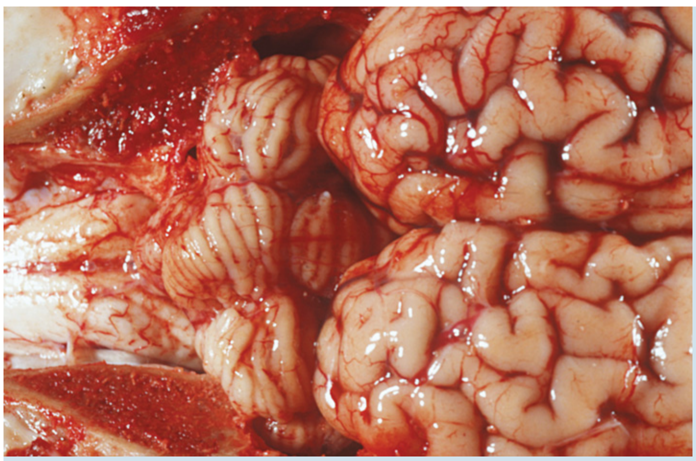

Figure 15.18: Cerebellar hypoplasia. Postmortem specimen showing a smaller than normal cerebellum.

Clinical signs

Signs including dysmetria and ataxia are evident as soon as the foal attempts to stand and walk.

Differential diagnosis

Ataxia at birth makes this distinguishable from other conditions such as cerebellar abiotrophy that develop later. Definitive diagnosis is only possible postmortem. The cerebellum is grossly and histopathologically abnormal.

Treatment

No treatment is possible but less severely affected animals may survive with a satisfactory quality of life. As it has not been shown to be an inherited condition continuing to breed from the parents is unlikely to result in the birth of another similarly affected foal.

Cerebellar abiotrophy

Abiotrophy means premature (postnatal) degeneration, and in the case of cerebellar abiotrophy refers to degeneration of neurons, particularly Purkinje cells, in the cerebellum. Cerebellar abiotrophy is a familial disease that occurs in purebred Arabian or Arabian crossbred horses in addition to Oldenburg, Gotland and Eriskay ponies.

History

Foals are normal at birth and up to a few weeks of age and then develop progressive clinical signs of dysmetria and tremoring.

Clinical signs

- Foals develop an ataxia gait with a hypermetric (high stepping) and stiff (hypometric) gait.
- The head and neck become progressively ataxic with wide, swinging head excursions, jerky head bobbing, an intention tremor and an abnormal menace response.
- Comparison with age-matched controls is useful as newborn foals have a gait suggestive of mild cerebellar dysfunction.

Differential diagnoses

- Cerebellar abiotrophy must be differentiated from other rare congenital tremor syndromes such as in utero exposure to organophosphates or Arnold–Chiari malformation, although these may also have other neurological signs.
- Acquired diseases such as abscessation and migrating parasites could be considered.

Diagnosis

- The diagnosis can only be confirmed at postmortem. A cerebellum to whole brain weight ratio of less than 10% confirms relative smallness of the cerebellum.
- Histopathology shows degeneration of Purkinje cells, atrophy of cerebellar folia and loss of the external granular layer.

Treatment

There are no treatment options and clinical signs can be expected to progress.

Juvenile epilepsy / Lavender foal syndrome (Figs 15.19 & 15.20)

History

Juvenile epilepsy occurs mainly in Egyptian Arabs or Arabian crosses. The disorder occurs in foals that are otherwise normal from several weeks to several months of age and unlike true epilepsy is usually outgrown.

Aside from the neurological signs, the most striking feature of some of these foals is the dilute or bleached-out hair coat color. In a few cases, the color is a very striking iridescent silver to pale lavender hue, hence the name "lavender foal syndrome". Coat color dilution lethal is a more appropriate name, as many affected foals do not exhibit the striking lavender color. Other dilute coat colors observed are pewter (pale slate gray) and pale chestnut (pink). Information obtained from one study strongly suggests a heritable condition that merits further investigation.

Clinical signs

Single or multiple epileptiform seizures. Frequently these seizures are not seen and all that is noticed is evidence of self-trauma. If seen, the seizures usually follow a consistent pattern which is individual for each foal.

Prodromal signs and post-ictal signs are also usually consistent for a given animal. Amaurosis (central blindness) is common before and after seizures and is frequently noted by owners.

Figure 15.19: This foal suffering from Lavender foal syndrome, had epileptic seizures starting at 3 months of age. Treatment consisted of a 12 mg/kg PO loading dose of phenobarbital followed by 6 mg/kg PO q24h for 2 months. The foal was 6 months of age and weaned when this image was taken. No further epileptic seizures had been noted and the medication was discontinued.

Diagnosis and differentials

The history (Arab or Arabian cross) and clinical signs are often suggestive of the diagnosis but all other possible causes of seizures in this age group should also be ruled out. These would include:

- meningitis
- other infectious agents such as strangles, viral encephalitis
- trauma
- metabolic disorders
- disorders of the cardiovascular system
- liver failure
- heat stroke
- fever resulting from infectious process elsewhere
- parasitic infection.

Treatment

- Although this disorder is out-grown, anticonvulsant therapy (phenobarbital) should be initiated at the first signs and maintained for several weeks to months.
  - The use of phenobarbital at a dose rate of 12 mg/kg PO/IV loading dose and then 6 mg/kg PO/IV q12h has been clinically rewarding. Phenobarbital induces microsomal enzymes, which may alter the metabolism of the drug with repeated administration. Serum phenobarbitol concentrations should be monitored and maintained between 10–30 μg/mL.
- Failure to treat these animals may result in neuronal death and further possibly permanent seizure foci.
- Underlying problems such as fever that may initiate further seizures should be corrected.

Figure 15.20: Arabian foal with juvenile epilepsy in the post-ictal phase. The foal was unaware of its surroundings and was blind for 10–15 minutes following the termination of the seizure. He was observed walking into walls and his dam at that time.

Hypoxic–ischemic encephalopathy (neonatal maladjustment syndrome) (Figs 15.21–15.27)

Hypoxic–ischemic encephalopathy (HIE) is a specific syndrome characterized by alterations in behavior, neurological signs and ischemic and/or hemorrhagic CNS lesions associated with perinatal asphyxia.

Pathophysiology

Hypoxia is the partial (hypoxemia) or complete (anoxia) lack of oxygen in the brain or blood. If the hypoxemia is severe enough, initially peripheral tissues and ultimately brain tissue will develop an oxygen debt, leading to anerobic glycolysis and the production of lactic acidosis. **Asphyxia** is the state in which placental or pulmonary gas exchange is compromised, or ceases, which typically progresses to hypoxemia. **Ischemia** is a reduction in or cessation of blood flow to an organ (brain), which compromises not only oxygen delivery to tissue but substrate delivery as well.

In utero, the fetus adapts to a relatively hypoxic environment by increased oxygen affinity of fetal hemoglobin, increased ability to extract oxygen from the blood and a greater tissue resistance to acidosis. Similar to the redistribution of blood flow in a diving seal, the severely asphyxiated fetus and neonate are able to redistribute oxygenated blood away from less vital organs (lungs, kidneys, skin and bowel) to more vital organs (heart, brain and adrenals). As a result of this protective mechanism, multiple organs may sustain injury. The equine fetus appears to have an oxygen demand "reserve" in that, under conditions of reduced oxygen availability, it decreases its rate of growth and decreases its oxygen consumption. This form of in utero growth retardation (IUGR) is termed disproportionate. The fetus is stunted and presents with a disproportionately large head, little muscle mass, small frail body and little to no fat. If the in utero asphyxia is severe, the fetus will not be able to sufficiently compensate and the CNS will be compromised. Many factors including gestational age of the fetus, severity of hypoxia and duration determine the severity of clinical signs and CNS lesions.

Adenosine triphosphate (ATP) is the primary energy modulator of all cells including neurons. In tissue hypoxia, ATP production by oxidative phosphorylation is curtailed, with concurrent increases in

Figure 15.21: Tongue protrusion in a foal with HIE.

Figure 15.22: Abnormal repetitive behaviour in a foal with HIE.

Figure 15.23: Trauma from seizures.

Figure 15.24: Chewing seizure in HIE foal.

Figure 15.25: Head pressing in a foal with HIE. Note the trauma evident on the forehead.

cellular adenosine disphosphate (ADP) and adenosine monophosphate (AMP). The loss of cellular ATP during hypoxia-ischemia severely compromises those metabolic processes that require energy for their completion. Thus, ATP-dependent sodium ion efflux through the plasma membrane in exchange for potassium is curtailed with a resultant intracellular accumulation of sodium and chloride as well as water (cytotoxic edema). Intracellular sodium and chloride ions and water continue to accumulate, resulting in alterations electrochemical gradients. Prolonged hypoxia-ischemia can also result in cell death of the capillary endothelium and tight junctions. Potentially this results in extracellular edema (vasogenic edema). How long a cell can survive in this situation is not known, as other factors that influence the ultimate cellular integrity are called into play. The role of extracellular edema and increased intracranial pressure in foals with HIE is still currently being debated, with no consensus at this time.

Figure 15.26: Paddling seizure in HIE foal.

The excitatory amino acid neurotransmitter glutamate also appears to be a major factor in the production of HIE injury. Under conditions of prolonged hypoxia-ischemia, neurons depolarize and leak glutamate, which cannot be removed rapidly from the extracellular space. Once activated, glutamate receptors activate the sodium and potassium ion channels. More critical however, is the influx of calcium that passes through glutamate-gated channels, especially the NMDA receptor. Calcium, in turn, sets in motion a cascade of biochemical events that cause the death of a neuron.

Excessively excited by high levels of glutamate, mitochondria – the main buffers of intracellular calcium – may become overloaded. The diminished mitochondrial function can lead to decreased energy to maintain ion gradients, potentially perpetuating a vicious cycle of membrane depolarization and NMDA receptor channel opening. The increased free cytosolic concentration of calcium activates numerous intracellular reactions that in excess can seriously compromise the viability of the neuron. These reactions include the activation of lipases, proteases and endonucleases, which disrupt the structural integrity of the cell. Calcium also contributes to the formation of oxygen free radicals via the formation of xanthine and prostaglandins.

History

Foals are often normal for the first few minutes to hours after birth and then may be unable to locate the udder or suck properly, lose affinity for the dam, wander aimlessly and can progress to having seizures.

The condition should be expected in any foal which has a history of abnormal placentation of the mare, premature placental separation, delivery by caesarian section, prolonged dystocia, premature delivery or in foals that have required resuscitation for any reason after delivery.

Clinical signs

- Clinical presentation is dependent on the degree of hypoxia.
- Mild hypoxia/ischemia results in a patient that may be stuporous, somnolent, lethargic and hypotonic.

Figure 15.27: Stringhalt in a neonatal foal. Note the exaggerated lifting of the hind leg. HIE may affect the central nervous system or may affect a single nerve resulting in bizarre presentations such as this one. Most often it is a diagnosis of history and exclusion in these cases and treatment is symptomatic. The foal in this image made a complete recovery after 3 days of treatment with intravenous DMSO.

- The patient may also have a loss of suckle reflex, dysphagia, odontoprisis, central blindness, mydriasis, anisocoria, nystagmus and head tilting.
- Patients who have been exposed to more moderate hypoxia/ischemia are more likely to experience seizures characterized by eye blinking, eye deviation, nystagmus, paddling movements and a variety of oral-buccal-lingual movements.

Differential diagnoses

- Metabolic disorders (e.g. hyponatremia)
- Hyperosmolality (hyperlipemia, hyperglycemia)
- Hepatoencephalopathy
- Infectious conditions
  - bacterial meningitis
  - bacteremia
  - EHV-1 meningitis
- Cranial trauma
- Development conditions
  - hydrocephalus
  - cerebellar abiotrophy.

Diagnosis

HIE is principally a diagnosis of exclusion. Foals typically present within 24–36 hours of birth. If cerebrospinal fluid (CSF) analysis is performed it sometimes reveals evidence of mild hemorrhage, however in most cases a spinal tap would not be recommended.

This is one area in which the use of atlanto-occipital ultrasound to diagnose increased CSF pressure may have a role.

Treatment

Treatment of HIE and the resultant success rates have improved significantly in the last 10 years; however many commonly used treatments are still quite controversial. The goals of treatment are listed below with many of the currently recommended treatments.

Prevention of intrauterine asphyxia

With increased awareness of fetal wellbeing and its impact on neonatal mortality, fetal monitoring has enabled the earlier identification of foals at risk of HIE. Early treatment of placentitis in mares may help to decrease intrauterine asphyxia.

Antimicrobials

Sulfonamide and trimethoprim combinations, gentamicin and penicillin are commonly used, as they have been shown to cross the placenta. Cultures of any vaginal discharge are also useful in guiding antimicrobial selection.

Tocolytics

Altrenogest (0.088 mg/kg PO q24h) is most commonly used and is thought to be clinically beneficial. Other possible tocolytics are clenbuterol and magnesium.

Pentoxifylline

Pentoxifylline is administered at a recommended dose rate of 7.5 mg/kg PO q8–12 h. This medication has both anti-inflammatory as well as rheological effects. A recent study has noted adequate concentrations in both placental and fetal tissues after pregnant mares were given a dosage of 8.5 mg/kg PO q12 h.

Antioxidants

Vitamin E (10,000 PO q24 h) is an antioxidant that is synergistic with ascorbic acid. While ascorbic acid is the principal antioxidant in the aqueous environment, vitamin E decreases the amount of lipid peroxidation and is the principal antioxidant in the lipid environment. The main problem with vitamin E is that it is lipid soluble; for an

effective dose to reach the brain or circulation, vitamin E needs to be given for some days before the ischemic insult.

Anti-inflammatories

NSAIDs are most commonly used.

Maintaining adequate ventilation

- It is important to avoid both hypoxia and hyperoxia which can lead to further neuronal injury.
- Hypercapnia may result in vasodilation, possibly leading to further hemorrhage.
- Monitoring should include pulse oximetry and expired CO_2. Arterial blood gases are advisable in many cases but proper handling of samples is important.
- There may also be a role for assisted or mechanical ventilation in many of these foals.
- Caffeine is used in many cases to improve respiratory drive resulting in improved ventilation.

Maintaining adequate perfusion

Vascular tone can be compromised following asphyxial injury characterized by hypotensive shock. Hypotensive shock can be treated with adequate crystalloid therapy and colloidal therapy.

- 1 L bolus of isotonic fluids followed by maintenance administration; 2–4 mL/kg/h of a low sodium-containing fluid such as 0.45% saline or 0.45% saline and 2.5 % dextrose.
- Plasma and hetastarch are suitable colloids. Plasma also offers beneficial immunoglobulins and other proteins which are often important in those foals with concurrent septicemia. Hetastarch is also thought to be beneficial in reducing reperfusion injury.

Maintaining adequate glucose levels

- Avoid hypoglycemia and hyperglycemia by maintaining glucose in the range 80–180 mg/dL.
- There is increasing interest in the use of insulin to maintain glucose in the normal range. It is now common practice in human ICU units to administer insulin to all patients on glucose-containing fluids.

Controlling seizures

Seizures lead to an increased cerebral metabolic rate which in turn leads to:

- a decrease in brain glucose
- a decrease in high energy phosphate compounds
- an increase in lactate
- excessive release of exitotoxic amino acids which lead to further neuronal injury.

Seizures also result in hypoventilation and apnea leading to hypoxemia and hypercapnia.

Seizures should be controlled with diazepam (0.1–0.2 mg/kg IV q15–30 minutes), midazolam (5 mg bolus then CRI 2–6 mg/foal/h) or phenytoin (2.8–16.5 mg/kg PO q8 h). If this is not adequate to control the seizures then phenobarbital administered as an initial loading dose of 8–16 mg/kg and followed by 5–10 mg/kg IV q8h can be used. Some practitioners dilute phenobarbital in saline but this is not necessary.

The effects of repeated doses of diazepam are not known but there may be a resultant decrease in SpO_2.

Control of cerebral edema and neuronal damage

- DMSO (0.25–1.0 g/kg IV q6–q12 hours as a 10% solution) is frequently used and benefits may be more related to its effect as a free radical scavenger and anti-inflammatory.
- Mannitol (0.25–1.0 g as a 20% solution q6–q12 hours as an IV bolus over 15–20 minutes) is also favored by many clinicians. If inappropriately dosed it may lead to osmotic diuresis and hypernatremia. Although it has been shown to decrease intracranial pressure in human neonates, it has not been shown to improve the clinical outcome and is not currently recommended for use in human neonates.
- Glucocorticoids have not been shown to have any beneficial effect and are not currently recommended in humans or equids.

Other treatments

Free radical scavengers

Allopurinol (xanthine oxidase inhibitor) 40 mg/kg PO within 2–3 hours of birth. It is inexpensive and there are anecdotal reports of its benefits.

NMDA (N-methyl-D-aspartate) receptor blockers

- Magnesium. 50 mg/kg IV infusion for 1st hour then 25 mg/kg/h CRI. There are mixed reports of the benefits or otherwise of magnesium. May not be effective if administered after the insult as would be the case with most foals.
- Ascorbic acid (vitamin C): 100 mg/kg/day IV. Inhibits neurotransmitter binding to NMDA receptors. In the fetus, ascorbic acid is one of the principal antioxidant systems. Plasma ascorbic acid concentrations in the brain are approximately 10-fold those in plasma. The optimal dosage of ascorbic acid for neuroprotection is not known. In high-risk human premature infants a dose of 100 mg/kg/day was found to be safe.

Thiamine

The usual dose is 1 g IV in 1 L of fluids SID. Thiamine increases the activity of the ATP-dependent sodium pump, thus regulating ion uptake and decreasing cellular water.

Hypothermia

This has been shown to be neuroprotective. However the exact level of hypothermia has not been established for equine patients but currently it is advisable to stay below rather than above normal temperature. Thus avoid overheating by the judicious use of heating lamps, blankets, etc.

- Good nursing care is vital (see Chapter 3, p. 76).

Prognosis

Without sepsis up to 80% of foals can survive. Prognosis is poorer for premature foals and foals showing clinical signs immediately after birth.

Hydrocephalus (Fig 15.28)

Hydrocephalus, an increase in cerebrospinal fluid volume within the ventricular system (internal hydrocephalus) or subarachnoid space (external hydrocephalus) is rare in horses. It is most often seen in neonatal foals as a congenital malformation.

An inherited defect has been proposed in some cases but the mode of inheritance has not been established. Hydrocephalus can also be acquired following conditions such as meningitis or cerebral hemorrhage.

Hydrocephalus can be classified as normotensive or hypertensive. Normotensive hydrocephalus usually is incidental to hypoplasia or loss of brain parenchyma after destructive prenatal or postnatal infection or injury. The CSF volume passively expands to fill the space that is normally occupied by the brain tissue.

Hypertensive hydrocephalus is a result of obstruction of the CSF conduit between the sites of production, in the third and lateral ventricles, and the sites of absorption by the arachnoid villi in the subarachnoid space. Blockage may be due to hypoplasia, or aplasia of a part of this system, or may be acquired. The increased CSF pressure results in dilation of the third and lateral ventricles with resulting tissue damage.

History

Foals with even severe congenital hydrocephalus may have no previous signs noticed by the owner and can suddenly be found dead.

Clinical signs

Foals may show nothing more than a somewhat domed head. Abnormal behavior including lack of recognition of the dam, compulsive walking, adopting bizarre postures and apparent blindness can be seen. Hydrocephalus can cause cerebral vasculature to be hemodynamically unstable and can result in acute death with minimal pre-mortem clinical signs.

Differential diagnoses

Hydrocephalus should be strongly suspected in foals presented for abnormal behavior with a domed forehead compared to age- and breed-matched controls.

Differentials for hydrocephalus are:

- Hypoxic ischemic encephalopathy
- Electrolyte imbalances
- Meningitis
- Hypoglycemia
- Premature weak foal
- Septicemia.

Diagnosis

Advanced imaging, specifically computed tomography and magnetic resonance imaging, are techniques which are increasingly available and can make a definitive diagnosis in the living foal. Postmortem examination of foals or fetuses while allowing a diagnosis seldom reveals the etiology.

Treatment

None currently available.

Narcolepsy / cataplexy (Fig 15.29)

Narcolepsy is a disorder of rapid eye movement (REM) sleep and is usually accompanied by cataplexy (profound loss of muscle tone). It is occasionally reported in foals and is familial in miniature horses and Suffolk horses.

History

Episodes of collapse in neonatal foals may occur spontaneously or be precipitated by a stimulus such as manipulation or, most commonly, feeding. The syndrome can be present from birth or can be precipitated after an intervention such as surgery.

Figure 15.28: Hydrocephalus in a foal showing prominent doming of the forehead.

Figure 15.29: Narcolepsy in an American miniature foal. This foal would show episodes of narcolepsy following feeding and was extremely difficult to arouse.

Clinical signs

There are no abnormalities between episodes. An attack may progress from buckling at the knees to sudden collapse with rapid eye movement, loss of skeletal muscle tone and absence of reflexes (e.g. patellar reflexes).

Some facial responses may be conserved. Cardiovascular function is normal. Foals can be aroused with varying degrees of difficulty.

The underlying etiology is unknown but may involve a defect in function of receptors of the neurotransmitter hypocretin (known to be involved in arousal and food intake in other species).

Differential diagnoses

Epilepsy and cardiovascular causes of collapse. These however are not associated with rapid eye movement or loss of reflexes.

Diagnosis

The diagnosis is made on clinical signs. Electroencephalography is used in other species but interpretation in foals requires considerable experience.

Treatment

There is no treatment but the condition resolves in some foals.

Figure 15.31: Violent seizures in a foal with meningitis. Note the blood on the walls from self-trauma. This foal was known to have fractured its skull following a kick. CSF analysis 3 days after the initial injury indicated a marked elevation in white cells. This foal recovered after prolonged antibiotic therapy.

Acquired conditions

Meningitis (Figs 15.30–15.33)

History

Foals may be presented for a stiff neck, blindness, changes in consciousness and seizures. Some foals have a history of failure of passive transfer, septicemia, sinus infections, cerebral wounds or coexisting illnesses such as enteritis.

Figure 15.30: Aimless wandering in a foal with meningitis.

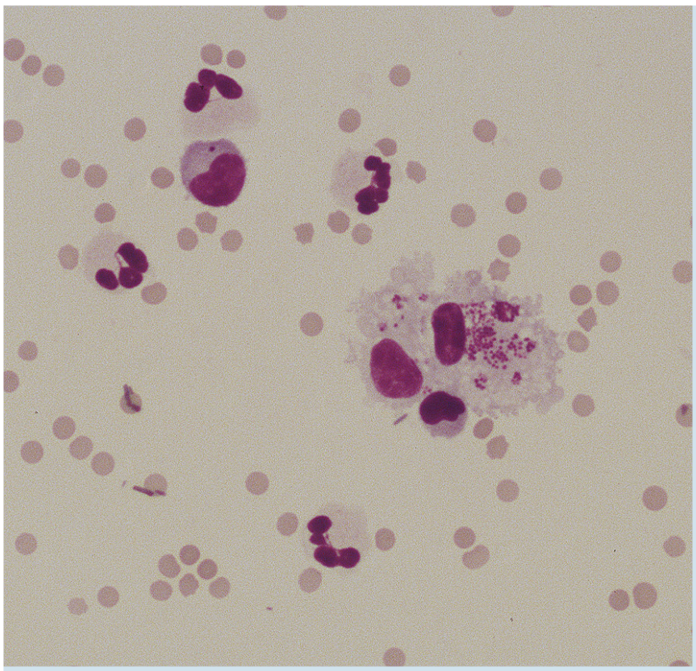

Figure 15.32: Septic meningitis. Cytology of CSF fluid demonstrates intracellular cocci and rods in a macrophage (Wright and Giemsa stain at 100×). Culture of the CSF sample yielded a growth of *Streptococcus zooepidemicus*. This 6-month-old QH foal was euthanized due to poor prognosis and financial constraints. The foal had presented with fever, cervical pain, and recumbency.

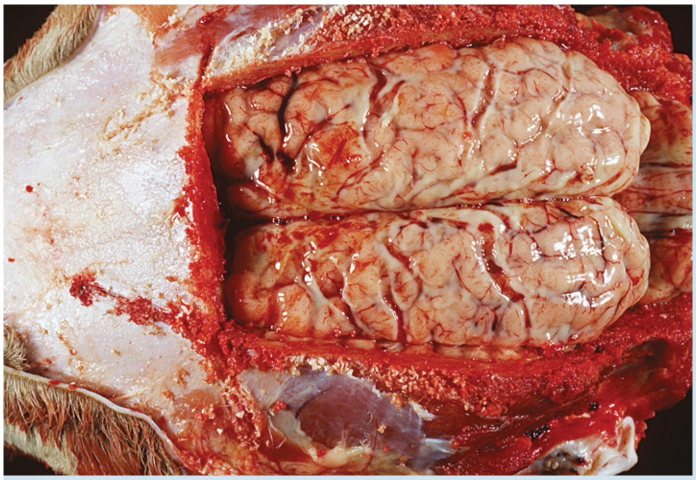

Figure 15.33: Postmortem specimen from a foal with septic meningitis. Note the purulent fibrinous exudate covering the cerebrum.

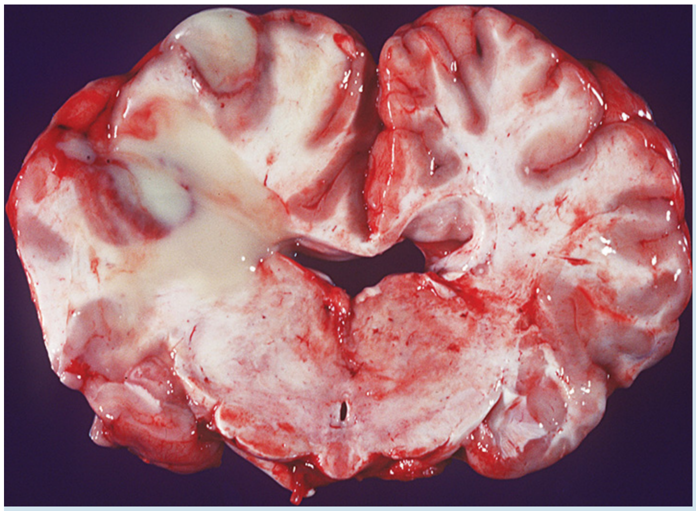

Figure 15.34: Multiple *Streptococcus equi* subsp. *equi* abscesses in the brain of a 7-month-old colt.

Clinical signs

A combination of fever, behavior changes, hyperesthesia, extended neck posture, and ataxia can be found. Some foals have shown concurrent omphalophlebitis or polyarthritis.

Differential diagnoses

Vertebral trauma or systemic illnesses resulting in "depression" must be ruled out.

Diagnosis

High white cell counts with neutrophilia can be expected on CSF analysis. Complete blood counts can show neutrophilic leukocytosis but sepsis frequently results in neutropenia. Blood cultures are worth pursuing. Coliforms, *Salmonella* spp and *Actinobacillus equuli* often are the causative organisms in foals. Viral, protozoal, fungal or immune-mediated meningitis is possible but much less likely.

Treatment

Four to six weeks of aggressive antibiotic therapy are indicated in cases of bacterial meningitis. If culture and sensitivity results are not available third-generation cephalosporins are a reasonable choice.

Cerebral abscess (Fig 15.34)

History

Cerebral abscesses may develop secondary to bacterial infections elsewhere in the body or via extension of a local disease process such as sinusitis, rhinitis, dental disease and periocular lesions. *Streptococcus equi* has been most commonly implicated, however a variety of species including *Strep. zooepidemicus, Rhodococcus equi, Actinobacillus equuli, Klebsiella* spp. and *Pasteurella* spp. may be involved. Bacterial meningitis may be present concurrently.

Clinical signs

- Signs become apparent with sufficient compression of cerebral tissue; the onset being insidious or acute depending on the rate of growth of the abscess. The clinical course is often characterized by marked fluctuations in the severity of signs.
- Behavioral changes such as depression, wandering or unprovoked excitement are most obvious.
- Contralateral impaired vision, deficient menace response and decreased facial sensation are consistent early findings.
- Affected horses frequently circle or stand with the head and neck turned toward the side of the lesion.
- Progression leads to recumbency, unconsciousness, seizures and signs of brainstem compression such as asymmetric pupils, ataxia and weakness.

Differential diagnoses

- Equine protozoal myeloencephalopathy (EPM)
- Trauma
- Viral encephalitis (West Nile virus infection)
- Portosystemic shunt
- Renal encephalopathy
- Rabies
- Electrolyte imbalances
- Hypoglycemia.

Diagnosis

- Ante-mortem diagnosis can be difficult. A history of recent or historical bacterial infection, particularly *S. equi* infection, should prompt further investigation of cerebral abscessation, although other causes of asymmetric cerebral disease should be ruled out.
- Hematology findings of hyperfibrinogenemia, hyperglobulinemia and leucocytosis may be present but are not consistent.
- Changes in the CSF depend on the degree of meningeal or ependymal involvement. Most cases exhibit xanthochromia and a moderate

elevation of CSF protein levels reflective of cerebral damage and compression.

- Computed tomography can be very useful for diagnosis but is currently of limited availability.
- The horse should be evaluated for the presence of a septic focus elsewhere in the body.
- Appropriate diagnostic specimens, including blood, should be submitted for bacterial culture in an attempt to identify the causative organism.

Treatment

If the signs are acute, severe and rapidly progressive, it is likely that brain edema is also present. The following medications have been used to counteract life-threatening increases in intracranial pressure (ICP).

- Corticosteroids (dexamethasone 0.1–0.5 mg/kg IV q24h).
- Osmotic agents
  - Mannitol 0.5–1 mg/kg as a 20% solution IV BOLUS over 15 minutes q24h to q12h
- Diuretics
  - Frusemide 0.75–1 mg/kg IV q24h to q12h.
  - DMSO 1 g/kg as a 10–20% solution q24h to q12h.

In addition to the treatment of ICP prolonged antibiotic administration would be warranted. Based on pathogen(s) isolated from cerebral abscesses in foals the use of the following would be of benefit:

- Potassium penicillin (22,000 to 40,000 IU/kg IV q6h) and gentamicin 6.6 mg/kg q24h would seem a good initial choice
- Third generation cephalosporins (cefotaxime or ceftazidime)
- Chloramphenicol
- Erythromycin, azithromycin or clarithromycin and rifampin if *R. equi* is suspected.

Aspiration of the lesion is potentially possible if the location of the lesion is known. However, in general the prognosis is poor and horses may be left with residual deficits.

Viral encephalitis

Alphaviruses and flaviviruses (Togaviridae) cause viral encephalitis in horses. The alphaviruses cause Eastern equine encephalitis (EEE), Western equine encephalitis (WEE) and Venezuelan equine encephalitis (VEE) while West Nile virus is now the most frequently encountered flavivirus. All are vector transmitted and, except for VEE, the horse is a dead-end host. Infections with these viruses are rare in Europe. Vaccination against EEE and WEE has decreased their incidence. Foals are susceptible to infection when maternal antibodies begin to wane.

EEE, WEE and VEE

Clinical signs reported include fever and generalized stiffness which is followed by ataxia, somnolence and proprioceptive defects. WEE is usually mild and rarely progresses beyond mild signs whereas EEE frequently results in severe signs and death.

Diagnosis

- Ante-mortem diagnosis may be difficult. Serological testing is often the preferred method. Antibodies rise rapidly after infection and are often present in 24 hours. A four-fold rise is diagnostic but may be difficult to demonstrate due to the acute rise and horses with EEE frequently do not live long enough to obtain paired titers. A single sample with an elevated neutralizing antibody, complement fixation or hemagglutination titer is very likely to be positive.
- When there is doubt as to whether a titer is the result of vaccination or disease comparing hemagglutination titers for WEE and EEE may be helpful; an eight-fold difference is considered diagnostic for EEE.
- IgM ELISA is available for VEE that can differentiate disease from vaccination titers.
- CSF analysis reveals leucocytosis and an elevated protein. Depending on the stage of disease neutrophils (early) or mononuclear cells (late) may predominate. CSF IgG and IgG index are elevated in most cases.
- Definitive diagnosis can be made at postmortem.

West Nile virus

West Nile virus is a mosquito-borne disease that affects a broad range of animals and causes a meningoencephalomyelitis. The progressive spread across the United States in recent years may be related to migration of avian reservoir hosts. Other than the United States, WNV is identified in many other countries including most of Europe, Africa, west and central Asia, Oceania and the Middle East. Many species can develop an immune response to the virus without demonstrating signs of disease.

Clinical signs

- Few infected horses show clinical signs.
- In those with signs, the onset is generally acute following an incubation period of 6–10 days. Foals from vaccinated mares will be protected by maternal antibody until approx. 6 months of age. Foals from unvaccinated mares could be affected at any age.
- A range of clinical syndromes exist from mild peripheral neuritis to encephalitis. Signs include pyrexia in approximately one-third of cases, ataxia which is most prominent in the hind limbs, muscle fasciculations and hypersensitivity to touch.
- Lethargy is associated with subclinical cases.
- Cases are most commonly seen in late summer to early fall in the US but might be observed year round in areas with prolonged vector seasons.
- No significant age or breed disposition has been identified.
- Euthanasia is warranted in cases that are unable to stand because of hindlimb paralysis or when signs of cerebral lesions (such as seizures or coma) are present.
- The majority that recover usually have a complete resolution of clinical signs in weeks to months.

Diagnosis

- WNV encephalomyelitis should be suspected in any horse showing signs of neurological disease in an area where WNV activity has been documented.
- Confirmation of a positive case of WNV encephalitis has been based on IgM-capture enzyme-linked immunosorbent assay (MAC-ELISA) of serum or CSF, plaque reduction neutralization test (PRNT) of serum, viral isolation and PCR performed on brain tissue.
- A MAC-ELISA revealing serum titers $\geq 1:400$ suggests recent exposure to the virus.

- High IgM titers decline within less than 2 months after exposure.
- PRNT with titers ≥1 : 10 indicates an IgG response but may not be present in the acute phase of the disease and may persist for 15 months
- Cytologic examination of the CSF shows a wide variety of changes in cell count and protein concentration.
- *Vaccination does not interfere with the ability to diagnose acute cases.*

Treatment and prevention

There is no specific treatment and therapy is based on supportive care. Prevention is also based on limiting exposure to mosquito vectors and on vaccination.

Cerebral trauma (Figs 15.35–15.40)

History

Trauma can be caused by running into a stationary object, kicks or falling over backwards onto the poll. Soft tissue injury may not be obvious in cases in which the traumatic episode was not observed.

Clinical signs

- Closed head injuries frequently result in fractures of the occipital, sphenoid and temporal bones and hemorrhages of the brainstem, venous sinuses and inner and middle ear cavities.
- Progressive obtundation, seizures, asymmetric pupillary light responses and central and peripheral blindness (the latter due to stretching of the optic nerves) may be observed.
- Unilateral vestibular signs (such as head tilt and nystagmus) are associated with petrous temporal bone fractures.
- Bilateral pupillary dilation and progressive loss of consciousness implies cerebral herniation and may warrant a grave prognosis.

Differential diagnoses

If a traumatic episode was not observed, severe cases of HIE may be a differential diagnosis in neonates.

Diagnosis

- Radiography or advanced imaging may be required in cases with skull fractures, discharge from ears or nose, or progressive clinical signs.
- CSF collection should be approached with caution due to the risk of herniation following the sudden release of increased intracranial pressure.

Treatment

- Patency of airways and treatment of hemorrhages and shock must be initially attended to.
- Foals with CNS signs following cranial trauma should probably receive dexamethasone (0.1–0.25 mg/gg q12–24h) for 1–4 days. It should be noted that the administration of dexamethasone for the treatment of supratentorial intracerebral hemorrhage in humans has failed to show a beneficial effect. Studies in humans with methylprednisolone (30 mg/kg bolus followed by constant rate infusion 4 mg/kg/h) have shown beneficial effects, which are thought to be derived from the stoichiometric antioxidant activity of the steroid.
- Intravenous hyperosmolar fluids such as mannitol (20%, at 0.25–1.0 g/kg over 20 minutes) or 7.5% hypertonic saline (administered at a dose of 4–8 mL/kg in 200–400 mL boluses) may be indicated to decrease cerebral edema.
- Surgical decompression may be attempted if bone fragments appear to penetrate the brain or the conscious state rapidly deteriorates.
- Supportive care with special attention to nursing is extremely important as some foals make remarkable long-term recoveries following cerebral trauma.
- $Alpha_2$ agonists (i.e. xylazine, detomidine) should be avoided in the acute stages as they can cause transient hypertension, exacerbating CNS hemorrhage, and can suppress ventilation.
- Interferon alpha given at 3 million units in 1 L of saline IV 1x daily for 3 days has been used with some success

Head tilt / facial nerve paresis (Figs 15.41–15.43)

History

Trauma to the back of the head may cause hemorrhage around the medulla or into the middle or inner ear. This may be complicated by fractures of the occipital and petrosal bones, separation of these bones or separation of the basioccipital and basisphenoid bones ventral to the pons and medulla. Preexisting osteoarthrosis and ankylosis of the temporohyoid joint(s), which is rare in young foals and may or may not be associated with otitis interna, predisposes to fractures of the hyoid bone and through the osseous bulla and adjacent petrous temporal bone.

The facial nerve (CN VII) innervates the muscles of facial expression. Facial paralysis is common in horses and depending on the site of damage some or all of the facial muscles may be affected. Damage to the proximal portion of the facial nerve is seen with fractures of the vertical ramus of the mandible, the stylohyoid bone or the petrous temporal bone. Other causes of unilateral facial paralysis without direct injury to the facial nerve are: medullary lesions involving the facial nucleus, polyneuritis equi, idiopathic facial paralysis, hemorrhage into the middle or inner ear, guttural pouch mycosis and parotid lymph node abscessation.

Clinical signs

- A careful neurological examination should be performed to determine the extent of vestibular and facial nerve involvement. The neurologic signs that result are often quite variable and asymmetric depending on which cranial nerves are affected and the extent of medullary parenchymal damage.
- Hemorrhage into the middle and inner ear cavities causes vestibular and facial nerve signs such as vestibular ataxia (a wide-based stance and staggering), a head tilt towards the affected side usually with an ipsilateral eye-drop (stand the foal square and lift the head, observe for disparity in the amount of visible dorsal sclera), circling towards the side of the lesion, ipsilateral facial paralysis and spontaneous horizontal or rotary nystagmus with the fast phase away from the side of the lesion.

Figure 15.35: Clearly demarcated "kick" to the side of the head of a 5-month-old foal showing neurological signs.

Figure 15.36: Fractured skull (fracture site is marked with an arrow).

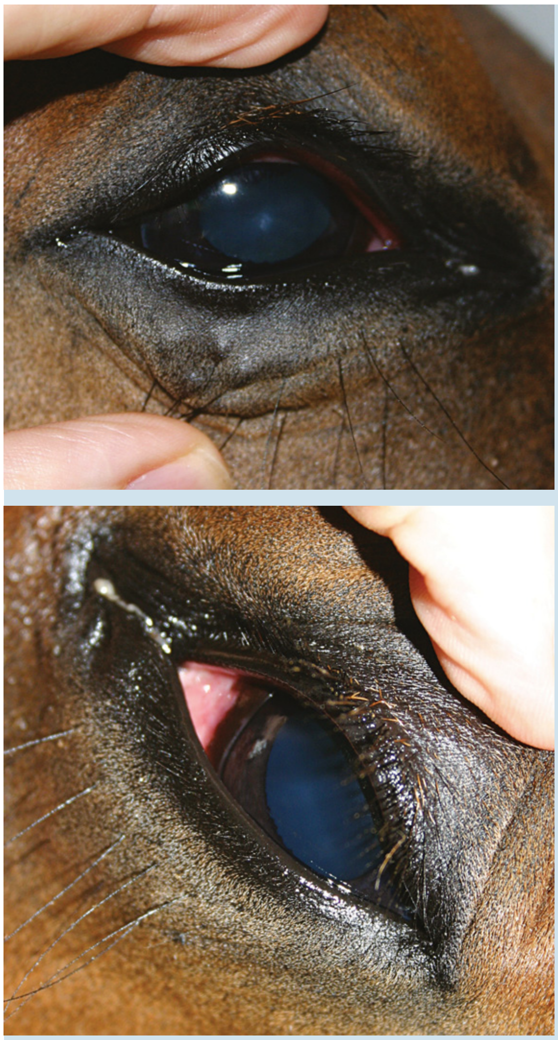

Figure 15.37: Bilateral pupillary dilation in a foal following head trauma (same foal as in Fig 15.35).

Figure 15.38: Same foal as in Fig 15.35. This image was taken 28 hours after the trauma occurred and shows an unconscious filly with "gasping" breathing, which is typically seen with severe brain edema and compromise of the respiratory centers. This foal died 30 minutes later.

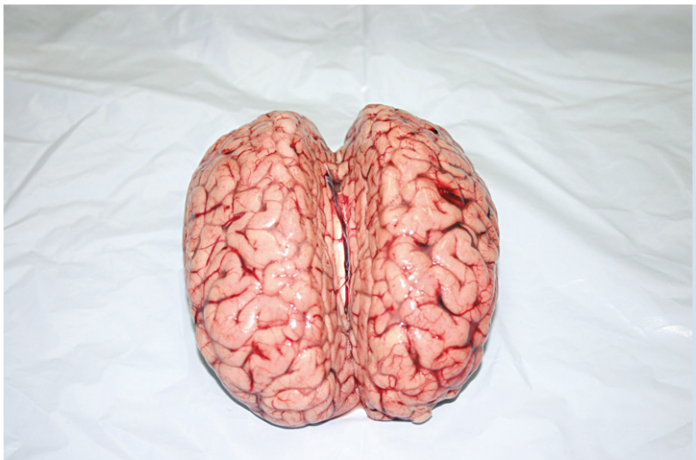

Figure 15.39: Same foal as in Fig 15.35, showing marked hemorrhages on the left side of the brain (right hemisphere in image) consistent with the area that was closest to the kick. No skull fractures were identified on postmortem examination.

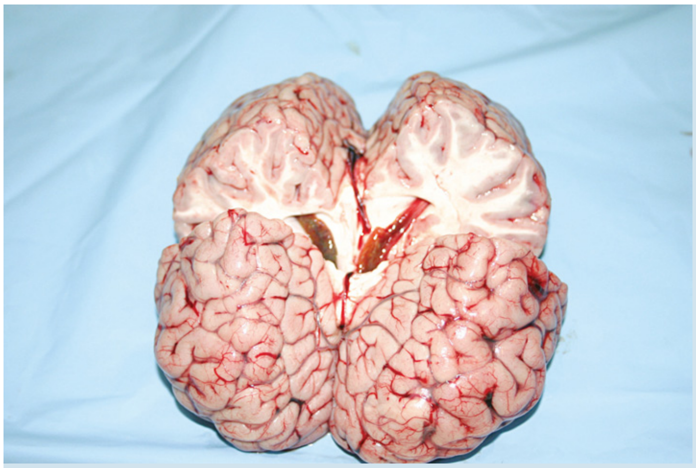

Figure 15.40: Same foal as in Fig 15.35, showing marked edema of the white matter of the left side of the brain (right hemisphere in image).

- With central or bilateral peripheral lesions (the latter rather rare) vertical nystagmus may be observed.
- A central lesion would further be expected to show other evidence of brainstem disease such as other cranial nerve signs, variable levels of obtundation and ataxia, paresis, or conscious proprioceptive deficits.
- Signs of facial nerve paresis vary with the location, severity, and chronicity of the lesion.
- Unilateral facial paralysis is more common and if located in the facial nucleus or proximal portion of the facial nerve is evident as deviation of the muzzle towards the normal side and drooping of the ipsilateral ear, eyelid and lip in addition to reduced flaring of the ipsilateral nostril during inspiration. Inability to close the eyelid causes exposure keratitis. There may also be damage to the secretomotor fibers of the facial nerve (parasympathetic portion) at or proximal to the geniculate ganglion. This results in reduced or absent tear production, which with eyelid paresis or paralysis can result in corneal ulceration. A Schirmer tear test can be used to determine if administration of artificial tears is needed.

Figure 15.41: Acute onset head tilt in a 2-month-old foal following head trauma. No fracture was evident on plain radiographs of the head. The foal recovered fully with supportive care.

Figure 15.42: Paralysis of the buccal branch of the facial nerve. On the right side (left when viewing the image) seen as a drooping of the nostril on the affected side.

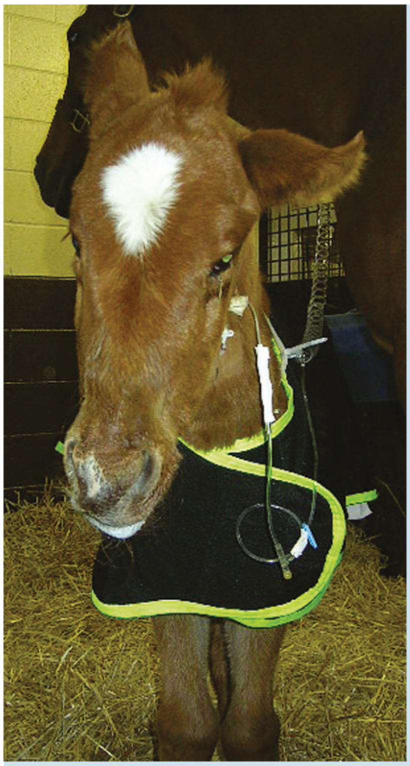

Figure 15.43: Marked facial nerve paralysis in a foal resulting in drooping of the ear, eyelid and nostril on the affected side, left side of the foal (right side when viewing the image).

- Bilateral facial nerve paralysis causes dysphagia, with dropping of feed and accumulation of feed between teeth and cheeks. It is important to determine the site of the lesion as it determines the prognosis.
- A lesion of the auriculopalpebral branch of the facial nerve, near the zygomatic arch, results in paresis or paralysis of the eyelids and ear only.
- A lesion of the palpebral branch of the facial nerve, crossing the zygomatic arch, results in paresis or paralysis of the eyelids only.
- A lesion of the buccal branch of the facial nerve, as it courses along the surface of the masseter muscles, results in paresis or paralysis of the lips and nostrils only.

Differential diagnoses and treatment

Trauma

- Trauma is a common cause of facial paralysis. Halter injuries and prolonged lateral recumbency may injure the buccal branches of the facial nerve on the side of the jaw and cause unilateral or bilateral paresis or paralysis of the lips and nostrils.
- A kick can result in fracture of the petrous temporal bone (which contains the middle and inner ear) resulting in vestibular and/or facial nerve signs.
- Electromyography, including electrical stimulation of the facial nerve, can be used to determine the location and severity of the injury.

Treatment for trauma or local inflammation consists of some or all of the following:

- NSAIDS (phenylbutazone 20 mg/kg IV, PO q12h or flunixin meglumine 1.1 mg/kg IV, PO q12h for 3–5 days) or systemic steroids (dexamethasone 0.05–0.1 mg/kg IV, PO q24h for 48–72 hours).
- The use of a topical anti-inflammatory cream such as 1% diclofenac sodium (Surpass™) may also be indicated to control inflammation associated with the facial nerve.
- The prognosis for the return of facial nerve function depends on the site and severity of the lesion. Without severe skin laceration, the prognosis for peripheral facial paralysis is good, although recovery may take several weeks to months.
  - If there have been no improvements after 6 months, the chance for recovery is poor.

Otitis media interna

- Otitis media interna although a common cause of facial paralysis and/or vestibular signs is rarely seen in foals.
- Otitis externa and a ruptured or diseased tympanic membrane may be seen on otoscopic examination under general anesthesia.
- Skull radiographs and magnetic resonance imaging or computed tomography may be necessary to confirm otitis media.
- The prognosis can be good, if the diagnosis is made early and the animal treated for 4–6 weeks with the appropriate antibiotic, determined by culture and sensitivity.
- The facial nerve paralysis can be permanent, and long-term administration of artificial tears may be necessary.

Spread from other sites

Guttural pouch infections can produce vestibular disease in horses by extension of the infection into the petrous temporal bone. Lesions of the facial nerve nucleus in the brainstem can result in facial nerve paralysis in equine protozoal myelitis and has been reported in a 2-month-old foal.

Sepsis of the temporohyoid joint in weanlings has been associated with hematogenous spread from other sites. These cases present with vestibular signs and facial nerve paralysis.

Vertebral / spinal trauma (Figs 15.44–15.49)

History

Acute onset of thoracic and pelvic limb dysfunction is seen. A traumatic episode may have been observed.

Clinical signs

Depending on the degree of compression the pelvic limbs may be affected more severely than the thoracic limbs and the foal may "dog sit," suggesting a thoracolumbar lesion. The tone and withdrawal responses of the thoracic limbs should be carefully assessed to determine if the lesion is cranial to the cervicothoracic intumescence. The site of evident soft tissue damage may not be directly related to the site of traumatic impact.

Figure 15.44: This foal presented with acute onset ataxia, regional sweating and an inability to move its head or neck, with a visually obvious deformity of the proximal cervical vertebrae.

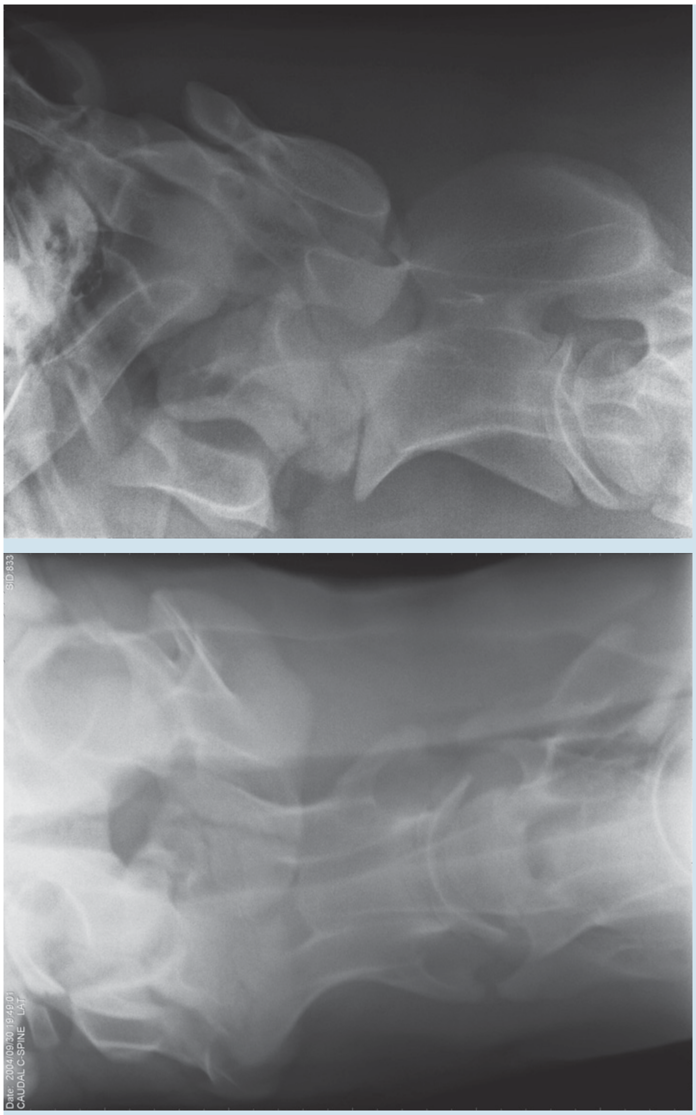

Figure 15.45: Same foal as Fig 15.44. Oblique lateral and dorsoventral radiographic views of the cranial cervical spine showing marked lateral malalignment of the vertebral canal and atlantoaxial articulation, consistent with atlantoaxial luxation. The head is facing left.

Figure 15.46: Weanling presented in recumbency with visually obvious deformity of the mid cervical area.

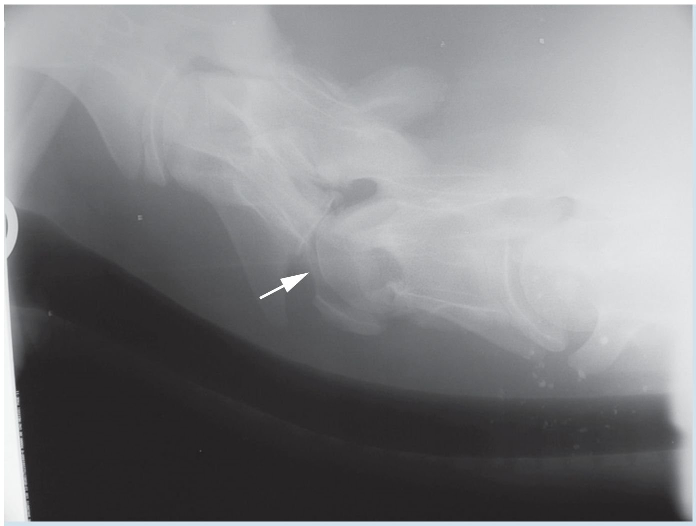

Figure 15.47: Lateral radiograph of the mid-cervical area of the foal in Fig 15.46. There is severe lordotic malalignment of the vertebral canal at C3–C4 due to fracture with distracted Salter–Harris type II physeal fracture of the caudal endplate of C3 (arrow).

Figure 15.48: A 4-month-old foal found collapsed in a field following an electrical storm. Physical examination revealed a step defect in the thoracolumbar spinal column (arrow).

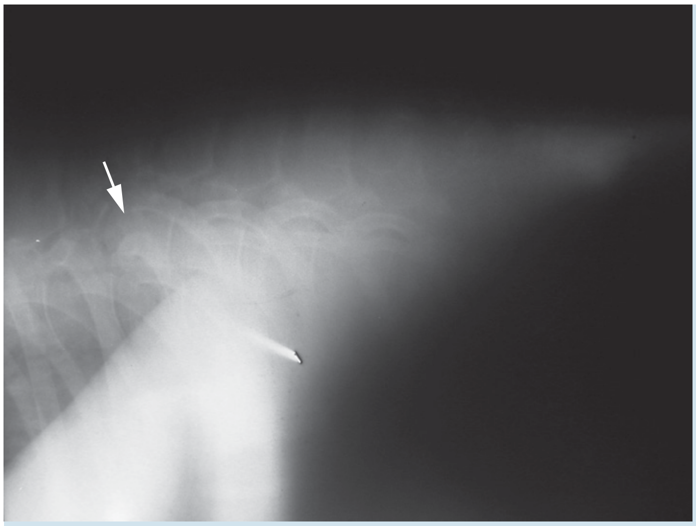

Figure 15.49: Lateral radiographic view of the caudal thoracic spine of the weanling in Fig 15.48. T10 is dorsally overriding the body of T9 (arrow) resulting in severe malalignment of the vertebral canal and indicating fracture-luxation of T9–T10. Note one rib appears to be overlying the other at the over-riding site (arrow).

Treatment

The decision to stabilize vertebral fractures must be taken in light of the severity of clinical signs and availability of specialist surgeons.

- It is arguable as to whether drug administration hastens recovery, but recommendations are similar to those for cerebral trauma with many authors recommending the administration of DMSO and dexamethasone.
- Pain should be managed with non-steroidal anti-inflammatories.
- However, caution should be used when administering analgesics or tranquilizers to ataxic patients as the animal may fall and worsen the lesion.
- Good nursing care is essential especially for recumbent patients and should include bladder and rectal evacuation if necessary.
- If the spinal fracture appears stable and the animal can stand with assistance, it may be placed in a sling and supported for long periods.
  - These should not be used for animals that cannot support themselves as severe respiratory compromise or myositis may result. Slinging of animals with mild neurologic signs may help minimize secondary complications, improve extensor tone and hasten recovery.

Thoracolumbar trauma

History

Acute onset pelvic limb dysfunction is seen. A traumatic episode may have been observed.

Clinical signs

- Degrees of pelvic limb paresis and ataxia can be evident. The foal should be carefully examined to determine that thoracic limbs are *not* involved (which may be not be obvious if signs are subtle).
- Cutaneous trunci reflexes may be lost caudal to a lesion if nerve roots are affected.
- If patellar reflexes and tone are normal the lesion is likely to be cranial to the L4 vertebra.
- Cauda equina signs may be found in cases of lumbosacral trauma.
- The site of evident soft tissue damage may not be directly related to the site of traumatic impact.

Differential diagnoses for cervical and thoracolumbar trauma

Space-occupying lesions such as abscesses similarly cause compression of the spinal cord. Parenchymal or subarachnoid hemorrhages are possible even in the absence of evident fractures and act as space-occupying lesions.

Diagnosis

- Every effort must be made to rule out fractures by radiography of the vertebral column in multiple oblique planes.
- Myelography or computed tomography may be required to rule out space-occupying lesions. However fractures may be made more unstable if muscle tone is reduced by inducing anesthesia.

Sacrococcygeal trauma

History

Acute onset cauda equina signs are seen. A traumatic episode may have been observed.

Clinical signs

The equine spinal cord ends in the L6 vertebra but the lumbosacral plexus is formed by the 4th through 7th lumbar nerves and all sacral nerves. Thus disease that affects any of the vertebrae from the caudal end of L4 through the sacrum may cause signs recognizable as motor or sensory abnormality in the nerves of the pelvic limbs and perineal region, autonomic functional abnormalities of the pelvic viscera, or both.

If the fracture site affects only sacral and cauda equina nerves, deficits in addition to pain can include a lack of detrusor activity resulting in a flaccid urinary bladder and overflow incontinence, a weak anus and paresthesia around the perineum. A hypotonic, flaccid, or analgesic tail may be noted.

Diagnosis

- Clinical diagnosis involves careful analysis of elicited history and correct interpretation of clinical neurologic examination.
- Lower motor neuron signs of the structures innervated by the cauda equina are useful, and exaggerated patella reflexes suggest involvement of the sciatic nerves (which normally antagonize the muscles innervated by the femoral nerves and dampen down the patella reflex).
- Pelvic limb gait abnormalities are frequently present in addition to pain on digital pressure over the sacrum and coccygeal areas.
- Radiographs of the caudal pelvis, sacrum and coccygeal areas are recommended but can be difficult to obtain.

- Nuclear images may be used when radiographs or an epidural contrast study are inconclusive.
- Electromyography of the perineal, paralumbar, and appendicular muscles can aid in localization of a lesion.

Treatment

- Medical management consisting of epidural corticosteroids (methyprednisolone acetate 200 mg diluted in 15 cc of distilled water) may result in clinical improvement.
  - The patient is sedated and the caudal epidural injection site is desensitized with lidocaine.
  - An 18 gauge spinal needle is then inserted into the epidural space and the medication injected.
  - Some patients may have to have the treatment repeated in 2–3 weeks, if pain to digital palpation over the sacral area is still present.
- The decision to stabilize sacral fractures must be taken in light of the severity of clinical signs and availability of specialist surgeons.

Peripheral nerves (Figs 15.50–15.52)

History

Peripheral nerve disease in foals is almost invariably due to traumatic lesions and the history indicates a sudden onset of non-progressive monoplegia/paresis.

Figure 15.50: Radial nerve paralysis in a foal secondary to a fracture of the humerus.

Clinical signs

- Selective sectioning of peripheral nerves can be expected to result in loss of sensation of the skin in autonomous zones.
- A foal presented with signs referable to one limb should have sensation of that limb systematically assessed using large artery hemostats – areas of hyposensitivity should be noted and the identity of the nerve subsequently compared with autonomous zones given in specialty text. Note that, unlike in small animals, the radial nerve does not have an autonomous zone in the horse.

Figure 15.51: Suprascapular nerve paralysis in a 7-month-old foal. The foal had been found markedly lame in the paddock 6 weeks previously. Pronounced lateral subluxation of the shoulder was evident on clinical examination. The foal was box rested for 4 months and then was turned out in a small paddock for a further 2 months before rejoining her herd mates.

Figure 15.52: One year after the initial injury the filly in Fig 15.51 is markedly improved with no gait abnormalities. This filly won her first start as a 2-year-old.

Differential diagnoses

Pain, for example a foot abscess or fracture, can result in clinical signs remarkably similar to loss of motor function.

Diagnosis

Once a painful focus has been ruled out the diagnosis is made on the basis of clinical signs. After 10–14 days disuse atrophy can be differentiated from neurogenic atrophy by the use of electromyography or a muscle biopsy. The three most common clinical syndromes are:

- **Suprascapular nerve paresis** ('Sweeney'): this is usually thought to be due to a traumatic injury of the shoulder secondary to a collision. Classic clinical signs are atrophy of the suprascapular and infrascapular nerves but the associated lateral subluxation of the shoulder may be due to additional involvement of pectoral and subscapular nerves or caudal cervical nerve roots.
- **Radial nerve paresis** (resulting in triceps paresis and lack of elbow extension): usually due to a brachial plexus avulsion secondary to extreme abduction of the affected thoracic limb. Careful neurological examination may reveal mild defects in other nerves of the brachial plexus.
- **Sciatic nerve paresis** is not infrequently affected by various injuries including intramuscular injections into the caudal thigh muscles. Clinical signs include poor limb flexion with stifle and hock extension and fetlock flexion when the foal is not weight-bearing on the limb. The leg can bear weight if the digits are extended since the stifle is extended by the femoral nerve (which is rarely affected in foals).

Treatment

Axons grow at a rate of only 1 mm per day and continue to do so for no more than 6–12 months before irreversible fibrosis sets in. Once neurogenic atrophy has been diagnosed the degree of atrophy should be monitored to help determine whether re-innervation has occurred and whether conservative treatment should be continued. Anti-inflammatories including dexamethasone 0.5 mg/kg IV and/or NSAIDs (phenylbutazone 2 mg/kg PO, IV SID-BID or flunixin meglumine 1 mg/kg PO or IV SID-BID) should be utilized to reduce perineural inflammation. Surgical release of fibrous entrapment may be considered after a few weeks.

Vertebral osteomyelitis (Fig 15.53)

Vertebral osteomyelitis (spondylitis) is an infectious or inflammatory degenerative disease of one or more vertebrae. Involvement of an adjacent intervertebral disc is termed discospondylitis. It has been related to hematogenous spread of infectious agents in the newborn and extension from local wounds. Progression of the vertebral infection leads to paravertebral abscess, meningitis, vertebral collapse and spinal cord compression. Many pathogens have been isolated including *Rhodococcus equi* in foals.

Clinical signs and diagnosis

- Fever, stiffness and sensory deficits with variable paresis are the signs that are usually first noted.
- The above signs are often preceded by undetected localized spinal pain. This may rapidly progress to recumbency.

Figure 15.53: Vertebral osteomyelitis of a coccygeal vertebra.

Diagnosis

Diagnosis is based on clinical signs, history, and positive findings on radiography, scintigraphy, computed tomography or ultrasonography. Hematology findings are usually consistent with inflammation and CSF analysis may be normal or consistent with spinal cord compression.

Treatment

- It is important to try to isolate the etiological agent prior to commencing therapy. Cultures of blood and feces should be performed in addition to cytology and culture of a fine needle aspirate.
- Other possibly useful tests should be borne in mind in animals with other clinical signs; for example tracheal wash cultures and cytology in foals suspected of *R. equi* infections.
- Long-term antibiotic therapy is required (3–6 months) and relapses are common. The success of treatment also depends on the severity of signs at presentation and intercurrent disease.

Cervical vertebral malformation / cervical vertebral stenotic myelopathy (Figs 15.54–15.56)

History

Cervical vertebral malformation (CVM) is a common cause of ataxia in horses and tends to affect young adults. It can however be seen in foals, and sometimes has a history of acute onset ataxia.

Figure 15.54: Cervical vertebral malformation in a 16-month-old colt; note the circumduction of the hindlimbs, truncal sway at the turn (right) and the hypermetric movement of the left forelimb (left).

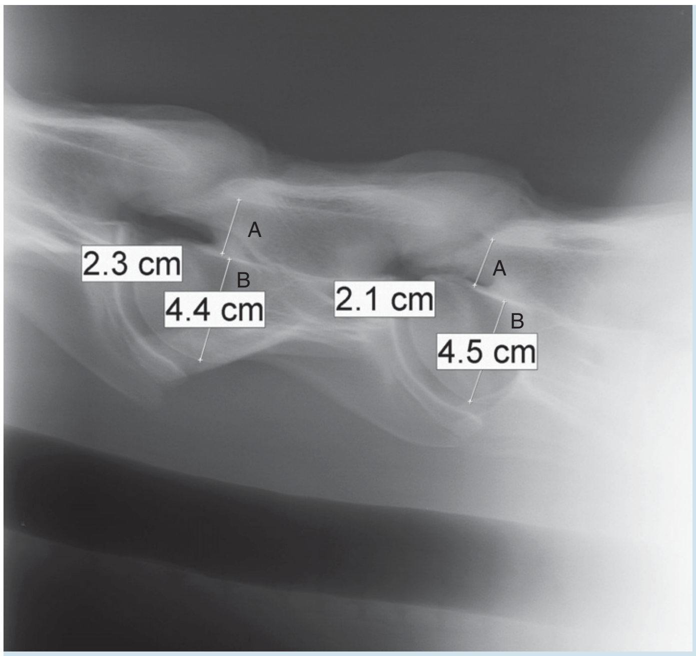

Figure 15.55: Minimum sagittal diameter measurements of the vertebral foramina. The vertebral foramen of C6 is small. There is a mild subluxation at C5–C6 with non-displaced osteochondral fragments of the articular processes and caudal epiphyseal flaring. The intravertebral sagittal ratio is A : B, thus at C5 is 52% and at C6 is 46%.

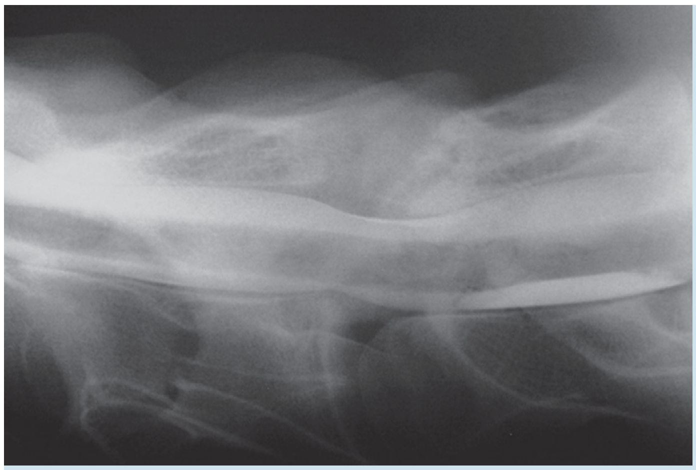

Figure 15.56: Lower cervical myelogram. There is approximately a 50% narrowing of the dorsal contrast column at C6–C7.

Table 15.1: System for grading of neurological gait deficits

GRADE	DESCRIPTION
0	No gait deficit
1	Deficit barely detectable at walk or trot, but present with special tests
2	Deficit detected during walk and trot, exaggerated by special tests
3	Deficit prominent at walk or trot, may fall during special tests
4	Stumbling, tripping or falling spontaneously at normal gait
5	Down, cannot rise

Clinical signs

- Compression of the cervical spinal cord results in lesions to proprioceptive and motor tracts to the thoracic and pelvic limbs. Table 15.1 outlines a system for grading neurological deficits that is useful in the evaluation of horses with suspected CVM as it allows for accurate recording and monitoring of progression.
- CVM results in ataxia (causing inconsistent foot placement and excessive circumduction of the pelvic limbs when turning the foal) and paresis (shown by weakness when pulling on the tail while the foal is walking in a straight line).
- Due to the peripheral location of pelvic limb tracts in the spinal cord they are affected more severely, resulting in paresis and ataxia which is more notable in the pelvic limbs. In mild cases the thoracic limbs will not appear to be affected even though the lesion is in the cervical cord.
- Currently two forms of CVM/CVSM are recognized – dynamic and static.
  - In horses with *dynamic stenosis*, compression of the spinal cord occurs only during movement of the neck, particularly flexion. Dynamic compression occurs most commonly at C3–C4 and C4–C5 and primarily affects horses at 8–18 months of age. Compression may not be evident on neutral radiographic views.
  - *Cervical static stenosis* is narrowing of the vertebral canal with subsequent compression of the spinal cord regardless of the position of the neck. Compression is evident on neutral radiographic views but in many cases is also exacerbated by extension of the neck, indicating coexisting dynamic compression. Static stenosis is most frequently seen at C5–C6 and C6–C7 and is usually seen in older animals, 1–4 years.

Differential diagnoses

Clinical signs of CVM can appear to be of acute onset and other causes of spinal cord ataxia, particularly vertebral fractures, need to be ruled out.

Diagnosis

Neurological examination usually reveals symmetrical ataxia, paresis, dysmetria and spasticity in all four limbs, though usually more noticeable in the pelvic limbs. Asymmetry of clinical signs can be seen in horses with significant degenerative joint disease of the articular processes. At a walk signs of ataxia and paresis such as truncal sway, circumduction of the hind limbs, toe-dragging and stumbling can be seen. These signs can be exacerbated by walking the horse up or down a slight slope, walking over obstacles (e.g. a curb), turning in circles or elevating the horse's head. Evidence of hypermetria, such as exaggerated limb movements, or hypometria, such as stiff-legged movements, are also frequently seen.

The diagnosis can be made from good quality cervical radiographs. Subjective assessment reveals enlarged physeal growth plates, caudal extension of the dorsal border of the orifice of the vertebral canal, angular fixation, delayed ossification of bone and degenerative joint disease.

Stenosis of the vertebral canal corrected for radiographic magnification can be determined by measurement of the intravertebral ratio. A ratio of less than 50% at C4, C5 or C6 or less than 52% at C7 is associated with a high likelihood of having CVSM.

Myelography can be used to confirm stenosis of the vertebral canal but should only be used when the results will alter the outcome or treatment of the case.

Contrast-enhanced computed tomography has been used in a few cases. Currently the indications are pre-surgical evaluation and assessment of lateral compressive lesions in horses without myelographic evidence of compression that are strongly suspected of having lesions based on clinical signs. Availability, cost and patient size are limiting factors for the use of this technique at present.

Treatment

- Although fusion of cervical vertebrae can result in resolution in clinical signs in selected adult cases of CVM, it would be hard to justify in a foal.
- Early diagnosis of CVM in young Thoroughbred horses has been successfully treated using a restricted PACE diet and confinement.
- The PACE diet is a low protein, low energy diet that has been implemented along with stall confinement on young Thoroughbred horses with cervical vertebral malformation/instability.
  - The best outcomes occur when foals are started at < 6 months of age.
  - A nutritionist should be consulted to design a ration that provides 65–75% of the National Research Council (USA) recommendations for crude protein and energy (total digestible nutrients).
  - The roughage provided is grass or low-quality timothy hay, with 6–8% protein.
  - The diet is formulated to ensure 100% of NRC levels of vitamins and trace minerals.
  - 5000 IU vitamin E is also supplemented to the foal.
  - The foal is maintained on the diet for 1 year with adjustments at 4–5 month intervals. The foal can be allowed access to small dry lots or a round pen.
  - In one study 75% of the foals with cervical vertebral instability were able to race after they completed the PACE diet. Animals on this diet will become very thin, which to some may seem inappropriate, but appears necessary.
- Treatment with anti-inflammatory drugs is also common. The use of corticosteroids is controversial due to lack of evidence regarding efficacy, potential negative side effects and the fact that corticosteroids are contraindicated in EPM. Without definitive diagnostic techniques differentiation between CVM and EPM in endemic areas may be difficult.

Equine protozoal myeloencephalitis

EPM is classically caused by a sarcocyst protozoa (*Sarcocystis neurona*) transmitted by the American opossum. Natural intermediate hosts include skunks, armadillos, raccoons and possibly cats. Horses are aberrant intermediate hosts.

The situation is complicated by the recent identification of a second parasite (*Neospora hughesi*) as a cause of EPM. The horse is an aberrant intermediate host of *S. neurona*, and the agent is able to produce a remarkable range of focal and multifocal neurologic signs.

Transmission of both *Sarcocystis* and *Neospora* parasites between mare and foal does not appear to occur during gestation. A vaccine is available but efficacy is as yet unproven.

History

EPM is only very rarely reported in young foals and epidemiological work indicates that major exposure probably does not occur on stud farms but happens later in the life of the horse, e.g. on the racetrack. Given the prevalence of EPM in some geographic regions it should probably nevertheless be considered if unexplained, often multifocal, neurologic signs occur in a foal. Clinical signs are often insidious in onset with a progressive course.

Clinical signs

The clinical signs of EPM result from the effects of inflammation, swelling and nerve cell death associated with protozoal merozoites and meronts in the CNS. The most common presenting complaint for EPM is ataxia, often with focal lower motor neuron signs such as gluteal atrophy, but can be any neurologic dysfunction.

Differential diagnoses

Most other diseases in this chapter should be considered. Diffuse cerebral signs would be unusual.

Diagnosis

- Antibodies acquired through ingestion of the mare's colostrum wane by approximately 3 months of age. These results indicate that foal sera might test positive for EPM in the first few months of life, even though they did not have contact with the EPM parasites.
- A blood sample can be submitted to a commercial laboratory (e.g. http://www.neogen.com/epm.htm) in order to test for anti-*Sarcocystis neurona* antibodies. This test has a poor positive predictive value (i.e. many horses have antibodies but do not have the disease) but a good negative predictive value (i.e. horses that are negative are highly unlikely to have EPM).

If the blood sample tests positive, submission of a CSF sample is appropriate. Care should be taken as even minor blood contamination can result in a false positive test.

Treatment

Currently there are three FDA-approved treatments for EPM:

- ponazuril (Marquis™, Bayer)
- 32% nitazoxanide (Navigator®, IDEXX) and
- sulfadiazine/pyrimethamine (ReBalance™, Phoenix Scientific).

Marquis and Navigator are paste formulations which are given once daily for 28 consecutive days and have demonstrated effectiveness in treating horses with EPM. Both of these products have a very favorable safety profile. ReBalance is a 1× daily oral antiprotozoal suspension with a usual treatment regime of 90–270 days. The dose rate in foals has not been established.

Equine degenerative myeloencephalopathy (EDM)

EDM is a diffuse degenerative disease of the spinal cord and caudal brainstem of young equids. It occurs in family lines in Thoroughbred and Morgan horses and has been reported in captive Przevalski's horse and Burchell's zebra.

The mode of inheritance has not yet been defined. Vitamin E deficiency has been implicated as having a role in the disease.

Ataxia and weakness, which may be quite sudden in onset, is reported. The condition usually affects horses under 6 months of age. Restricted access to vitamin E (green grass!) is in the history of some cases.

Clinical signs

Usually there is symmetric ataxia and weakness, with the pelvic limbs often affected far more severely than the thoracic limbs. Local skin reflexes over the neck and trunk may be reduced.

Differential diagnoses

Other conditions affecting the spinal cord of foals, such as CVM and trauma, should be excluded.

Diagnosis

- The diagnosis is by exclusion.
- Definitive diagnosis is made on *postmortem* examination by the recognition of degeneration of specific nuclei and axons in the spinal cord and caudal brainstem.
- Low serum vitamin E concentrations (<1.0 μg/mL) may be found in some (but not all) recently affected cases.

Treatment

Supplementation with 6000 IU vitamin E orally per day in addition to ample forage may improve the clinical signs. Affected foals are unlikely to improve completely.

Botulism ("shaker foal" disease) (Figs 15.57–15.60)

Horses are highly sensitive to the toxin produced by the *Clostridium botulinum* toxin. Eight toxin types have been identified but those most commonly associated with disease are Types A, B and C. Type A has been reported in California. The highest incidence in the United States is seen in Kentucky and the mid-Atlantic region and is generally associated with Type B toxin.

There are two main forms of botulism. Toxicoinfectious botulism, also known as "shaker-foal syndrome" occurs almost exclusively in foals as a result of overgrowth of *C. botulinum* in the intestinal tract

Figure 15.57: Botulism in a foal. Pharyngeal paresis has resulted in an inability to swallow. Note the milk coming from the nostrils soon after nursing.

Figure 15.58: Botulism foal on a ventilator. This foal required assisted ventilation for 10 days and then was gradually weaned off the ventilator. Such foals often require extensive physiotherapy and assistance in rising for a number of weeks after removal from the ventilator.

following the ingestion of spores and resulting in the by-production of neurotoxins. The disease most often affects fast-growing foals from 1–2 months of age but can occur up to 8 months of age. The mature, protective gastrointestinal microflora of adult horses typically prevents overgrowth of *C. botulinum* following ingestion. Signs are usually seen 1–4 days after ingestion of spores with the affected animal becoming recumbent within a week and dying shortly thereafter. More than one foal may be affected on the premises.

Figure 15.59: Close up of attachment of ventilator tubing to endotracheal tube. Intensive nursing care is required for botulism foals on ventilators.

Figure 15.60: Foal in a sling. The use of slings can be beneficial in rehabilitating botulism foals, treating milder cases of botulism or for rehabilitation following any neurological disorder that has resulted in prolonged recumbency.

In adult horses, botulism occurs following ingestion of pre-formed toxins in feed. Spoiled hay or silage are most commonly implicated in botulism caused by types A and B. Silage with a pH greater than 4.5 is favorable for sporulation and toxin production. This is known as "forage poisoning". It has also been suggested that birds may be able to carry preformed toxin from carrion to the feed of horses.

Type C botulism is associated with ingestion of feed or water contaminated by the carcass of a rodent or other small animal. Less commonly, botulism can occur when neurotoxins are produced in wounds infected with *C. botulinum*. Proliferation of *C. botulinum* type B organisms in gastric ulcers, foci of hepatic necrosis, abscesses in the navel or lungs, and wounds in skin and muscle have been associated with toxicoinfectious botulism.

The disease is characterized by progressive muscular weakness caused by the action of botulism neurotoxin at cholinergic

neuromuscular junctions. Botulinum neurotoxin has also been linked to equine grass sickness.

Clinical signs

- Affected foals are often found lying down and unable to rise.
- If seen early, the foal may lie down more than usual, often with its head resting on the ground, and if forced to walk, may have decreased limb flexion and toe scuffing. Generalized muscle tremors develop, hence the name "shaker foal."
- Dysphagia due to pharyngeal and esophageal dysfunction is common.
- Left untreated, the disease can be rapidly fatal, with death occurring secondary to respiratory muscle paralysis within 24–72 hours of the onset of clinical signs.
- Diffuse lower motor neurological signs such as depressed reflexes (including PLRs), decreased eyelid tone, decreased limb and tongue tone may be found on neurological examination.

Differential diagnoses

Other causes of weakness in foals need to be ruled out, particularly sepsis, hypoglycemia, colic, pneumonia and white muscle disease.

Diagnosis

This is a diagnosis by exclusion. Definitive diagnosis is very difficult as horses are more sensitive to the clostridium toxin than either mouse or commercial ELISA tests!

Treatment

- Very mildly affected foals can survive with minimal treatment.
- Immediate treatment with a polyvalent antitoxin prevents binding of the toxin to presynaptic membranes. However, antitoxin cannot reactivate neuromuscular junctions that have already been affected. Thus, antitoxin administration may have little effect in animals that are severely affected. Generally, only one dose of 200 mL of antiserum to foals (30,000 IU) of antitoxin is needed and provides passive protection for up to 2 months.
- Antibiotics should be administered if toxicoinfectious botulism is suspected or if there are secondary lesions such as aspiration pneumonia or decubital ulcers. Antibiotics that can cause neuromuscular blockade and possibly exacerbate clinical signs such as aminoglycosides should be avoided and neurostimulants such as neostigmine should not be used.
- Good nursing care including the provision of a deep bed and a quiet environment are essential. Frequent turning of recumbent animals, nasogastric feeding and fluid support for animals with pharyngeal and lingual paralysis, frequent catheterization of the urinary bladder, application of ophthalmic ointments and ventilatory support may all be required.
- A survival rate of 88% has been reported in foals with toxicoinfectious botulism that were provided with intensive nursing care (including mechanical ventilation and botulism antitoxin). However, this type of treatment is not available in all areas and is quite expensive. Without aggressive supportive care, the mortality rate is high, with death usually occurring 1–3 days after the onset of clinical signs.

Tetanus

Tetanus is caused by the toxin released by anerobic *Clostridium tetani* bacteria – horses are exquisitely sensitive to the toxin. *C. tetani* is normally found in the feces of horses.

History

The organism cannot grow in normal tissue but flourishes in deep, penetrating wounds and occasionally superficial wounds. If the mare and foal have been correctly vaccinated (foals should be vaccinated at 3 and 4 months of age, and then annually; broodmares should be vaccinated one month prior to foaling), then tetanus is extremely unlikely.

The action of the toxin is to inhibit the production of the inhibitory neurotransmitter glycine at CNS interneurons. Toxin binding is irreversible. The incubation period is 1–3 weeks.

Clinical signs

The disease is characterized by increased muscle tone and specifically prolapse of third eyelid, spasm of facial and masticatory muscles, pricked ears, and exaggerated responses to external stimuli such as noise. The mortality rate can reach 80%.

Differential diagnoses

Initial signs can be subtle, and could resemble a gait deficit for orthopedic reasons. Other reasons for myotonia are possible but extremely rare. Sequential clinical examinations should suggest tetanus early in the course of disease.

Diagnosis

Due to the extreme sensitivity of equids to tetanus, the disease has to be diagnosed on the basis of clinical signs.

Treatment

- Treatment is based on the knowledge that the toxin-ganglioside bond is irreversible and that gradual replacement of altered gangliosides by normal metabolic processes will lead to recovery. With this in mind, treatment is primarily symptomatic and supportive.
- The primary objectives of therapy are
    - destruction of *C. tetani* organism
    - neutralization of unbound toxin
    - control of muscle spasm
    - general supportive care.
- Neutralization of unbound toxin involves administration of large amounts of tetanus antitoxin (TAT). The recommended doses and frequency of administration for TAT vary widely. The dose that is given should be based on the history of the case and influenced by such factors as delay in treatment of several hours after injury, lack of aggressive debridement or no history of vaccination. Recommended doses range from 5000–20,000 IU intravenously and/or 5000–20,000 IU intrathecally. Intrathecal administration of tetanus antitoxin has been used with variable success.
- In addition to TAT administration, tetanus toxoid should also be administered as protective humoral immunity is not induced by natural disease

- Penicillin (sodium penicillin 20,000–40,000 IU/kg IV q6h or procaine penicillin 20000–40000 IU/kg IM q12h) should be administered.
- Wound care, including debridement, is important.
- Sedation (acepromazine 0.05–0.1 mg/kg IM or IV q4–6h) may be useful to reduce anxiety and provide muscle relaxation. Muscle-relaxing drugs such as guaifenesin should not be used.
- Supportive care is important. Horses should be placed in a dark, quiet environment with minimal stimulation. Ear plugs may be useful. Intravenous fluid therapy will be required in dysphagic horses. Nutritional care, including feeding via a nasogastric tube or parenteral nutrition, may be necessary. Frequent passage of a nasogastric tube should be avoided to reduce stimuli.
- Horses which are presented in lateral recumbency have little chance of recovery and euthanasia should be considered.

Rabies (Fig 15.61)

Rabies is a predominantly fatal neurological disease caused by a lyssavirus (*Rhabdoviridae*). The presentation can be highly variable and should be considered as a differential in any horse showing neurological signs in an endemic area.

Domestic animals which are generally regarded as dead-end hosts may become infected as a result of contact with wildlife vectors. Rabies is most commonly transmitted by salivary contamination of a bite wound, although infection by inhalation, oral or transplacental routes has been demonstrated in some species.

The incubation period varies from 2 weeks to several months depending on the site of inoculation, dose and pathogenicity of the viral strain. Following inoculation the rabies virus replicates locally and after several days attaches to peripheral nerve receptors and is passed from there to the CNS.

The cause of death may be respiratory paralysis as a result of infection of the medulla. Shedding of the virus in nasal and salivary secretions has been shown to predate the onset of clinical signs by up to 29 days.

Figure 15.61: Rabies in a weanling colt. Note the marked salivation.

Clinical signs

- The presenting signs and clinical course are extremely variable. Reported signs have included any of the following: anorexia, depression, blindness, mania, hyperesthesia, muscle twitching, lameness, paresis, ataxia, colic, urinary incontinence and sudden death.
- The most commonly reported signs in horses are hyperesthesia and recumbency.
- The disease is normally rapidly progressive once signs are seen and results in death in 3–10 days.
- Differentiation of rabies from other encephalitides on the basis of clinical signs is impossible.

Diagnosis

- Rabies has many differentials including other conditions with signs of gray matter disease such as polyneuritis equi, herpes virus myeloencephalitis, protozoal myeloencephalitis and sorghum–Sudan grass poisoning.
- Other differentials include cerebral diseases such as hepato-encephalopathy, leukoencephalomalacia, alphavirus encephalitides, occupying masses and meningitis.
- CSF findings are usually non-specific and can include moderate increases in protein, mononuclear cells and occasionally neutrophils.
- The gold standard for diagnosis is an indirect fluorescent antibody test using slices of brain which accurately diagnoses 98% of clinical cases.
- Microscopic examination of hematoxylin-and-eosin stained brain sections may reveal non-suppurative encephalitis and negri bodies which are diagnostic.
- Intracerebral inoculation of mice is also considered an accurate method of diagnosis.

Treatment

- Other than recovery in a presumptive case of experimentally produced rabies in a donkey, the disease has been reported as invariably fatal.
- In the rare situation where an ante-mortem diagnosis is reached the animal should be euthanized to avoid further human contact.
- Transmission from horses to humans has never been reported but should none the less be regarded as a possibility and all necessary precautions should be taken when dealing with animals demonstrating neurologic signs in an endemic area.

Prevention

- Foals in endemic areas should be vaccinated at 4–6 months of age with two doses given 3–4 weeks apart and followed by a booster at one year of age.

- The disease must be reported in the United States, Canada and many other countries and managed in conjunction with public health officials.
- If a previously immunized animal is bitten by a suspected rabid animal, it can be given three booster immunizations over 1 week and quarantined for at least 90 days.
- Exposed, unvaccinated animals of low economic value should be euthanized immediately.
- If the animal is valuable, confinement and close observation for at least 6 months is necessary. Primary immunization can be administered 1 month before release from quarantine.

Recommended reading

Donawick WJ et al 1990 Early diagnosis of cervical vertebral malformation in young Thoroughbred horses and successful treatment with restricted, paced diet and confinement. Proceedings of the Annual Convention of the American Association of Equine Practitioners 35:525–528

Mayhew IG 1989 Large animal neurology: a handbook for veterinary clinicians. Lea and Febiger, Philadelphia, 1989

Mayhew IG et al 1993 Diagnosis and prediction of cervical vertebral malformation in Thoroughbred foals based on semi-quantitative radiographic indicators. Equine Veterinary Journal 25(5):435–440

Nout YS, Reed SM 2003 Cervical vertebral stenotic myelopathy. Equine Veterinary Education 15(4):212–223

Foal admission assessment

Patient ______________________________

Farm ______________________________

Admission date ______________________ Admitting vet ______________________

Presented for ______________________________

HISTORY

Mare

Age:_____# of previous foals:_____ Problems with previous foals?_____no _____yes (details) ______________________________

Uterine infections/vaginal discharge?_____no _____yes ______________________

Illness during pregnancy?_____no _____yes ______________________

Milk dripping?_____no _____yes How long? ______________________

Last vaccinated?_____no _____yes Dates/What? ______________________________

Last deworming?_____no _____yes When/What? ______________________

Feeding: ______________________

Breeding date:________________ Foaling date: ______________________

Duration of pregnancy:____________term?_________ early ________overdue (_____days)

Dystocia?_____no _____yes (describe)______________________________

Premature placental separation *(red bag delivery)*? _____no_____yes ______________________

Early cord rupture? _____no _____yes ______________________

Entire placenta passed? _____no _____yes _____Condition of placenta? ______________________

Meconium staining of placenta? _____no _____yes ______________________

Udder:_____normal _____abnormal ______________________

Colostrum quality:_____normal _____poor _____quantity _____normal _____reduced ______________________

Foal

Spontaneous breathing? _____no _____yes ______________________

Time to stand? ______________________

Time to nurse? ______________________

Meconium stained? _____no _____yes ______________________

Nursing normally?_____no _____yes ______________________

Behavior normal? _____no _____yes (specifics) ______________________

Colostrum/milk given? _____no _____yes ________Amount/When ______________________

How colostrum given _____bottle _____tube _____other (syringe) ______________________

IgG tested? _____no _____yes __________Plasma given? ______________________

Urination? _____no _____yes _____Umbilicus treated? _____no _____yes ______________________

Meconium passed? _____no _____yes _____Enema given? _____no _____yes _____# given ______________________

Presenting complaint: ______________________________

Medications/treatments? _____no _____yes______________________

(specifics)______________________________

Medicine clinician______________________ Name______________________ Owner______________________

Patient ____

Farm ____

Admission date ____________________ Admitting vet ____________________

Presented for ____

Physical examination______________________ **Date:**__________ **Time:**________ **am / pm**

Temperature:______˚F Heart rate:_____/min Respiratory rate:_____/min Body wt:_____

Observation

Behavior: ____

Signs of prematurity?_____no _____yes (_____haircoat _____forehead _____ears _____joints_____tendons)

Skin/Body condition: ____

Suckle reflex:_____good _____moderate _____weak _____none ____________________

Eyes:_____normal _____Entropion (___L___R) _____Uveitis (___L ___R)_____Corneal ulcer (___L___R)

Sclera ____________________L____________RPLR normal/slow/absent

Cardiovascular

Pulse quality:____strong ____moderate____weak ____regular ____irregular ____________________

Mucous membranes:___pink____pale____injected____cyanotic____toxic____other____ CRT:____sec

Skin turgor:_____normal _____abnormal ____

Jugular veins:____normal____collapsed____distended____ Catheter____left____right

Cardiac auscultation:HR:_____ Intensity:_____ Rhythm:_____ regular_____ irregular

Murmur?_____ no_____ yes (describe)____

Respiration

Respiratory effort?_____normal_____decreased_____increased_____other ____________________

Nasal discharge?_____no _____yes____________________cough _____no _____yes ____________________

Auscultation:_____normal _____ abnormal (describe) ____________________

GI tract

Nursing?_____no _____yes IF NOT, pain related? _____no _____yes ____________________

Colic_____no _____yes Level of pain _____mild _____moderate _____severe

Distended abdomen _____no _____yes GI sounds?:_____LV _____LD ______RV _____RD

Meconium? _____no _____yes Fecal consistency: ____________________

Cleft palate _____no _____yes

Urogenital

Umbilicus:_____normal _____abnormal ____________________

Urination:_____no _____yes _____straining ____________________

Scrotum/testes-vulva/vagina_____normal _____abnormal ____________________

Musculoskeletal

Joints:_____normal _____abnormal ____________________

Lameness? _____no _____yes __________Angular limb deformities? _____no _____yes ____________________

Neurologic

_____normal ______abnormal ____________________

Seizures:_____no _____yes

Other? ____

____

Attending medicine clinician ____________________ **Attending intern** ____________________

APPENDIX 2 Foaling record sheet

Name of mare:

Foaling details Foaling box number:________

Last service date:___/___/___

Expected foaling date:___/___/___

Actual foaling date:___/___/___ Gestational age:_________

Time of first contraction:__________ Time of membrane rupture:________

Time feet seen:__________ Time of foal delivery:________

Was assistance required? YES/NO

Comments:____

____

Previous history of dystocia/foal illness:

____

Mare details

Was the mare stitched? Y/N/Don't know

Was there vulval laceration? Y/N/Don't know

Maternal acceptance of foal:____

Colostrum quantity and quality:____

Did the mare run milk before foaling? Y/N/Don't know

Was the mare sick before foaling? Y/N/Don't know

Placenta

Time placenta passed: ___________ (time after foaling:_______mins/hrs)

Placenta weight:______kg

Placenta appearance: Edema: Y/N Hemorrhage: Y/N Avillous areas: Y/N

Comments:

Foal details

Color:_________ Sex: C/F Weight:______kg Mare weight:_______kg

Breed:________________________ Sire:__________________________

Was the foal stained with meconium? Y/N

JFA test performed? Y/N Result: Pos/Neg

Time of delivery:_________Time to rise:_________ (time after delivery: mins/hrs)

Time to suckle:___________ (time after delivery: mins/hrs)

Enema given: Y/N (time after delivery: mins/hrs)

Time meconium passed: __________(time after delivery: mins/hrs)

Time of urination:________________(time after delivery: mins/hrs)

Colostrum fed? Y/N Amount:__________ Source:_____________

Time colostrum was fed:__________ (time after delivery: mins/hrs)

Plasma given? Y/N Amount: Type:

Umbilicus treated with:____

Additional foal details

Coat: long/short Silky: Y/N Floppy ears: Y/N Entropion: Y/N

Conformation:

	RF	LF	RH	LH
Flexor laxity:	none/mild/severe	none/mild/severe	none/mild/severe	none/mild/severe
Contracted tendons:	none/mild/severe	none/mild/severe	none/mild/severe	none/mild/severe

Angular deformities:

Comments:

APPENDIX 3 Reference values for hematology

CBC	Units	< 24 hours*	From birth**
WBC	K/μL	6.38–14.2	5.0–12.6
RBC	M/μL	7.13–12.01	6.5–9.99
Hemoglobin	g/dL	10.72–16.10	11.0–16.0
Hematocrit	%	31.02–48.36	33.0–48.0
Platelet count	K/μL	122–316	115–450
MCV	*f*L	37–46	38–53
MCH	pg		12–16
MCHC	g/dL	29–39	31–37
Differential			
Neutrophil %	K/μL	5.06–12.09	2.75–8.19
Lymphocytes %	K/μL	0.08–1.88	1.75–5.67
Monocytes %	K/μL	0.27–1.07	0.00–0.75
Eosinophil %	K/μL	0.02–0.09	0.00–0.38
Basophil %	K/μL	0.00–0.06	0.00–0.25
Fibrinogen			200–500
TPP			5.0–7.0

*Source: Janadriyah Farm, values derived from 61 normal foals within 24 hours of birth. All CBC analyses were performed on an Idexx analyzer in 2006.
**Source: Hagyard Equine Medical Institute, Clinical Laboratory. Normal reference values for equines from birth.

Alterations in total WBC values

Leukocytosis can be the result of physiologic causes (stress, excitation, exercise, corticosteroid administration) or can be pathologic. Physiologic leukocytosis is transient with neutrophilia and lymphocytosis.

Leukopenia is always pathologic. Foals with a total WBC <5000 cells/μL and an absolute neutrophil count <1000 cells/μL, with one or more risk factors should be considered candidates for antimicrobial therapy.

Alterations in neutrophil values

Bacterial infection is the most common cause of pathologic neutrophilia. A left shift (increased immature neutrophils in peripheral blood) may be seen in the acute stages of an infection.

A degenerative left shift (generally associated with neutropenia) is when the number of immature neutrophils exceeds the number of mature neutrophils and is a poor prognostic indicator.

Neutropenia is usually caused by bacterial septicemia and endotoxemia. Neutropenia that persists for greater than 4 days may be an indicator of inadequate granulopeiesis.

Rebound neutophilia often follows neutropenia associated with endotoxemia and is a good prognostic indicator.

Alterations in lymphocyte values

Pathologic lymphocytosis is uncommon but may be seen with chronic viral infections and autoimmune disorders. Physiologic lymphocytosis is common in young horses.

Lymphopenia is most common seen with acute viral diseases but will also be seen with such disorders as endotoxemia, septicemia and certain immunodeficiencies.

APPENDIX 4 Reference values for biochemistry

Test	Units	<1 week of age	2–3 weeks of age
Albumin	g/dL	2.7–3.6	3.4–4.1
Alkaline phosphatase (ALP)	U/L	1247–2853	25–871
Aspartate aminotransferase (AST)	U/L	70–194	127–399
Blood urea nitrogen (BUN)	mg/dL	14.8–28	11–26
CO_2	mEq/L	25.3–34.0	25–32
Calcium	mg/dL	10.6–14.52	11.6–13.2
Creatinine	mg/dL	1.3–3.2	0.8–1.8
Triglycerides	mg/dL	9.9–30.4	6–54
Total protein	g/dL	1.7–7.0	6.0–7.9
Lactate dehydrogenase (LDH)	U/L	307–479	176–477
Phosphate	mg/dL	4.6–6.8	1.3–6.0
Glucose	mg/dL	93.8–185.6	76–119
Gamma-glutamyltransferase (GGT)	IU/L	10.9–53.7	3–38
Chloride	mEq/L	91.6–110.5	97–105
Potassium	mEq/L	3.2–4.8	2.5–5.0
Sodium	mEq/L	133–147	133–140
Creatine kinase (CK)	U/L	90–1518	67–377
Direct bilirubin	mg/dL	0.11–0.48	0.7–2.5
Magnesium	mg/dL	1.36–3.01	1.4–2.3
Lactate	mmol/L	0.67–5.74*	0.9–1.65
Bile acids	µmol/L	0–30	0–30
Ammonia	mg/dL	0–63	0–63

Source: Hagyard Equine Medical institute, Clinical Laboratory. The values given for foals <1 week of age were derived from serum chemistry analysis performed on 70 healthy foals.

*Lactate was measured within 24 hours of birth.

Alkaline phosphatase (ALP) isoenzymes are found in every tissue with high activities in liver, bone, intestine, kidney and placenta. Neonates can be expected to have values of 100× that of adults in the first 10 days of life, especially shortly after the ingestion of colostrum.

γ-Glutamyltransferase (GGT) activity in foals can be noted to be significantly increased over that in the adult horse for the first month of life. GGT activities in the foal are not the result of colostric absorption, but primarily due to endogenous sources. The ratio of hepatic weight to total body weight is greatest in young foals and decreases with age. This disparity in relative hepatic mass may account for some or all of the increases in GGT values in foals. Human fetal hepatic tissue is reported to have specific activities of GGT 10× higher than those in adults. (AJVR 1986, Vol 47:11)

Formulary

Drug	Indication	Amount, route, frequency
Acepromazine	Sedative. Suggested sedative for use in botulism cases	0.01–0.05 single IV 0.02–0.10 single IM 0.03–0.15 single PO
Acetazolamide	Treatment for hyperkalemic periodic paralysis	3 mg/kg PO for treatment of episode of HYPP 2–4 mg/kg q8–12 h as control
Acetylcysteine sol. 4%	Enema for meconium impaction	200 mL PR, through Foley catheter and clamp for 20 minutes prior to evacuating rectum
Acyclovir	Anti-viral for herpes	10 mg/kg IV q12 h over 60 minutes. 20 mg/kg PO q6–12 h
Albuterol	Bronchodilator	0.01 mg/kg PO q8–12 h
Allopurinol	Xanthine oxidase inhibitor (used to prevent free radical formation in foals at high risk of developing HIE)	40 mg/kg PO within 2–3 hours of birth
Altrenogest	Treatment of uteroplacental inflammation	0.088 mg/kg PO q24 h
Amikacin sulphate	Antibiotic (aminoglycoside)	22–25 mg/kg IV q24 h for neonates 15 mg/kg IV q24 h for older foals and adults
Aminocaproic acid	Antifibrinolytic, blocks plasminogen	20 g in 1 L saline over 1 h, then 10 g in fluids q6 h
Aminophylline	Bronchodilator, diuretic	5 mg/kg IV in fluids q6–12 h as a diuretic. 5–15 mg/kg in fluids q6–12 h as a bronchodilator
Amoxicillin	Antibiotic	10–30 mg/kg PO q8–12 h
Ampicillin sodium	Antibiotic	10–30 mg/kg IV q8–12 h
Aspirin	Anti-platelet, anti-thrombotic	Adults: 7–10 mg/kg PO q24–48 h
Atropine sulphate	Bronchodilator, parasympatholytic	0.02–0.10 mg/kg IV, IM or SQ (bronchodilation). 1 mg/90 kg IV (parasympatholytic)
Aurothioglucose, i.e. gold salts	For pemphigus that is non-responsive to steroids	50 mg IM test dose; then 1 mg/kg IM weekly; decrease from weekly to monthly treatments
Azathioprine	Immunosuppressant, may use in place of steroids	3 mg/kg PO q24 h
Azithromycin	Antibiotic (macrolide), used in the treatment of Rhodococcus	10 mg/kg q24 h for 5 days then q48 h (*caution: may cause hyperthermia in foals*)
Barium sulfate	Given once as anti-diarrheal	3 mg/kg PO

Drug	Indication	Amount, route, frequency
Bethanechol	GI prokinetic, enhances urinary bladder and sphincter tone	0.07 mg/kg SQ q8 h to enhance bladder tone. 0.3–0.4 mg/kg PO q6–8 h or 0.025 mg/kg SC q6–8 h as a GI prokinetic
Bismuth subsalicylate	Anti-diarrheal	1 oz (30 mL) per 100 kg body weight PO q6–8 h
Butorphanol	Analgesic/sedative	0.05–0.1 mg/kg IV or IM
Cabergoline	To suppress lactation	2–3 mg PO q12–24 h in 500 kg mare
Caffeine	Respiratory stimulant for "dummy foals"	Loading dose: 10 mg/kg PO Maintenance: 2.5 mg/kg PO q12 h
Carprofen	NSAID	0.7 mg/kg q12–24 h PO
Cefazolin	Antibiotic (first generation cephalosporin)	11–20 mg/kg q6-8 h IV/IM
Cefepime	Antibiotic (fourth generation cephalosporin)	11 mg/kg IV q8 h
Cefquinome	Cephalosporin (fourth generation)	1 mg/kg IV or IM q24 h
Ceftaxime	Antibiotic (third generation cephalosporin)	20–40 mg/kg IV q6 h
Ceftazidime	Antibiotic (third generation cephalosporin)	20–50 mg/kg IV q6–12 h
Ceftiofur	Antibiotic (third generation cephalosporin)	2.5–10 mg/kg IV, IM or SQ q6–24 h (can be irritant if given IM or SQ)
Ceftriaxone	Antibiotic (third generation cephalosporin)	25–50 mg/kg IV or IM BID
Cephalexin	Antibiotic (first generation cephalosporin)	5–25 mg/kg IV or 5–20 mg/kg PO q4 h
Cephalothin	Antibiotic (first generation cephalosporin)	11–18 mg/kg IV or IM q8 h
Chloramphenicol palmitate (oral)	Antibiotic	44–50 mg/kg PO q6–8 h
Chloramphenicol sodium succinate (parenteral)	Antibiotic	25 mg/kg IV or IM q4–6 h
Cimetidine	H_2 antagonist: anti-ulcer medication	15 mg/kg PO or 6.6 mg/kg IV q6–8 h
Ciprofloxacin	Fluoroquinolone antibiotic; may destroy cartilage in foals	
Clarithromycin	Antibiotic (macrolide), used with rifampin to treat *R. equi*	7.5 mg/kg PO q12 h with rifampin
Clenbuterol	Bronchodilator; tocolytic (blocks uterine contractions)	0.8 µg/kg PO q12 h as bronchodilator 0.8 µg/kg PO as required of the oral preparation or 3 mg total dose IV of the injectable preparation as a tocolytic
Codeine phosphate	Anti-diarrheal	0.25 mg/kg PO q4 h

Drug	Indication	Amount, route, frequency
Colchicine	Inhibition of liver cirrhosis	0.03 mg/kg q12–24 h
Dantrolene	Post-anaesthetic myopathy. Myositis/tying up	2–4 mg/kg PO q24 h
Dapsone	Useful adjunct to traditional therapy (potentiated sulfas) of *Pneumocystis carinii* pneumonia. Treatment is usually prolonged 45–50 days	
Detomidine	Sedative	0.005–0.04 mg/kg once IV or IM
Dexamethasone	Anti-inflammatory, immunosuppressant	0.05–0.2 mg/kg once or q24 h IV or PO
Dextran	Colloid alternative to hetastarch	4 mL/kg IV
Diazepam	Sedative and anti-convulsant	0.11–0.44 mg/kg IV, may repeat after 30 min if required (i.e. 5–15 mg to a 50 kg foal)
Dihydrostreptomycin	Antibiotic treatment for Leptospirosis	25 mg/kg IV q24 h
Dipyrone	Anti-pyretic, analgesic	22 mg/kg IV or IM
DMSO	Decreases CNS, spinal and pulmonary edema. Anti-inflammatory, free radical scavenger	1 g/kg as a 20% solution in isotonic fluids given over 30 mins, q12–24 h for 3–5 days
Dobutamine	Cardiac inotrope for heart failure, shock	5–10 μg/kg/min as IV infusion
Dopamine	Potentially increases cardiac output and renal perfusion	1–5 μg/kg/min as IV infusion
Domperidone	Treatment for agalactia and prolonged gestation	1.1 mg/kg PO q12–24 h. Should be given 10 days prior to expected foaling date for mares known to have been grazing fescue pastures
Doxapram	Respiratory stimulant	0.5 mg/kg IV
Doxycycline	Antibiotic	10 mg/kg PO q12 h
Enalapril	ACE inhibitor; treatment of congestive heart failure	1 mg/kg PO q24 h
Enilconazole	Anti-fungal	0.2% solution for topical application used in the treatment of dermatophytosis
Enrofloxacin	Antibiotic (fluoroquinolone) – *use with caution in foals as it can cause cartilage damage*	7.5 mg/kg PO or 6 mg/kg IV q24 h
Epinephrine	Cardiac inotrope for anaphylaxis/ cardiac standstill, may administer via the trachea in foals	0.01–0.03 mg/kg (0.5–1.5 mL of the 1 : 100 solution for a 50 kg foal), every 3 minutes
Erythromycin	Antibiotic (macrolide) for treatment of *R. equi*	15–25 mg/kg PO q8 h

Drug	Indication	Amount, route, frequency
Eye solution	Subjective anti-infective for treatment of corneal ulcers/ hypopyon	1 g ancef, 5 cc geticin, 4 cc LA atropine, 10 cc diflucan in 50 cc saline administered by MILA pump
Famotidine	H_2 antagonist, anti-ulcer medication	2–3 mg/kg PO BID
Fenbendazole	Larvicidal anthelminthic (benzimadazole), fibrinolytic, anti-ulcer	10 mg/kg PO SID for 5 days
Fentanyl patch	Narcotic analgesic for pain control	100 mcg/h per patch; 1 patch for up to 120–340 kg. Clip hair but do not scrub site. Change patch q2–3 days
Ferric hyaluronate gel	Prevention of adhesions in surgery	
Fluconazole (Diflucan®)	Anti-fungal	10 mg/kg PO loading dose followed by 5 mg/kg q24 h for systemic fungal infections. 2 mg/kg loading dose followed by 1 mg/kg q24 h maintenance dose (oral candidiasis)
Flunixin meglumine	NSAID	0.25–1.5 mg/kg q8–24 h IV
Folic acid	Supplement for EPM therapy	400–500 mg/day
Furosemide	Diuretic	0.25–2 mg/kg q1–4 h
Gabapentin	Decreases neuroexcitability and neurologic pain	13–19 mg/kg PO q12–24 h
Gentamicin	Antibiotic (aminoglycoside), nephrotoxic	6.6 mg/kg IV or IM q24 h
Griseofulvin	Anti-fungal for dermatophytic skin infections	10 mg/kg PO q24 h for 7 days
Heparin	Anticoagulant; anti-thrombotic, lipase activator for fatty liver syndrome	40–80 IU/kg SQ or IV q8 h or 80–100 IU/kg in IV drip q6 h
Heparin (low molecular weight)	Anti-inflammatory; anti-thrombotic	Dalteparin: 50–100 IU/kg SQ q24 h Enoxaparin: 40–80 IU/kg SQ q24 h
Hyaluronate sodium	Treatment for non-infectious synovitis	40 mg IV weekly for 4 treatments
Hydralazine	Reduces cardiac afterload	0.5–1.5 mg/kg PO q12 h
Hypertonic saline	Shock treatment	2–4 mL/kg as IV bolus
Imipenem–Cilastatin	Antibiotic (potentiated penicillin)	5 mg/kg IV over 20 mins, q6–8 h
Ibuprofen	Non-steroidal anti-inflammatory	Up to 25 mg/kg q8–12 h for 6 days
Imipramine HCl	Treatment of narcolepsy	1.0–1.5 mg/kg PO q12 h
Insulin	Hormone for regulation of blood insulin	Protamine zinc: 0.15 U/kg q12 h IM or SQ or 0.1 U/kg SC as needed. Regular: 0.00125–0.2 U/kg/h IV CRI (pre-treat infusion lines as insulin adsorbs to lines)
Interferon alfa	Allergic airway disease, small airway inflammation	Human: 50 IU PO q24 h for 5 days Recombinant: 90 IU PO q24 h for 5 days

Drug	Indication	Amount, route, frequency
Iohexol	Diagnostic agent for equine myelography	20–40 mL into subarachnoid space (to replace volume of CSF removed)
Itraconazole (topical sol.)	Anti-fungal	1% in 30% DMSO
Itraconazole	Treatment for Aspergillosis (expensive)	6 mg/kg PO q12 h
Ivermectin	Anthelminthic	200 µg/kg once PO
Ketamine		1.0–2.5 once IV
Ketoconazole	Anti-fungal and blocks thromboxane	10 mg/kg PO q12–24 h (BID for *Aspergillus* spp)
Ketoprofen	NSAID	2.2 mg/kg IV q24 h for 5 days
Lactase	Enzyme replacement in foals with lactose intolerance	1500–3000 IU q4–12 h
Lactulose	Decreases ammonia production in liver disease	0.4 mL/kg PO q6–12 h
Levamisole	Immune modulator	2–3 mg/kg PO for 3–5 days
Lidocaine	Prokinetic for ileus	1.3 mg/kg slow bolus over 5–15 minutes, then 0.05 mg/kg/min CRI
Lufenuron	Fungal keratitis	5 mg/kg PO q24 h
Magnesium sulphate	Maladjusted foals	50 mg/kg IV infusion for first hour then 25 mg/kg/h IV slowly as CRI
Mannan oligosaccharides (Biomos®)	Gastrointestinal disturbances secondary to *Salmonella* or *Clostridium* spp	
Mannitol	Diuretic, reduces CNS edema, anti-oxidant	0.5–1 g/kg IV over 1–2 h as a diuretic q6–12 h. 0.5–1 g/kg as a 20% solution IV slowly over 30 min to reduce CNS edema q6–12 h
Methocarbamol	Muscle relaxant, used for myositis, tying-up and muscle spasms	5–25 mg/kg IV slowly, 25–75 mg/kg PO q12 h
Metoclopramide	Stimulates gastric contraction and GI motility in foals, rapid infusion may cause CNS stimulation	0.15 mg/kg SQ q6 h 0.1–0.2 q8–12 h slowly IV or PO
Methylprednisolone	Corticosteroid	1–2 mg/kg IV once
Metrizamide	Diagnostic agent for myelography	9.33 g in 21 mL saline/250 kg
Metronidazole	Antibiotic (anaerobic infections)	10–15 mg/kg PO q8–12 h 10 mg/kg PO q12 h for foals < 5 months
Midazolam	Sedative for use in neonates especially during mechanical ventilation	5 mg bolus, then 2–6 mg/kg/h CRI
Misoprostol	Prostaglandin E analog with GI anti-secretory and mucosal protective properties.	3–5 µg/kg PO q12–24 h

Drug	Indication	Amount, route, frequency
Naloxone	Opioid antagonist: Treatment for post-foaling hemorrhage Used for "dummy" foals, especially after C-sections to help "wake" them up	10–20 mg IV once 4 mg per foal IV once
N-butylscoplammonium bromide (Buscopan™)	Spasmolytic. Good for meconium impactions before the use of an acetylcysteine enema. Also useful for spasmolytic colic seen before the onset of diarrhea	0.3 mg/kg once IV
Nebulization solution	Treatment of muco-restrictive airway disease	3–10 mL of 20% acetylcysteine solution with 8 mL $NaHCO_3$ and 42 mL H_2O
Neomycin sulphate	Antibiotic (aminoglycoside). Gut sterilization in liver failure	5–15 mg/kg PO q24 h
Neostigmine	Stimulates large colon motility, used to treat tympanitic foals	Foal: 1 mg SQ q1–2 h
Neupogen® (Filigrastim)	Granulocyte stimulating factor. Used for foals with persistent luekopenia	300 µg SQ per foal. Frequency of administration varies with response. Response should be seen in 24–48 hours
Omeprazole	Proton blocker. Anti-ulcer medication	4 mg/kg PO q24 h for 2–4 weeks then 1 mg/kg SID maintenance
Oxacillin	Penicillinase resistant used for treatment of Staph. infections. Often used for osteomyelitis	20 mg/kg IV or IM q6–8 h
Oxyglobin	Artificial hemoglobin for oxygen transportation	5–30 mL/kg at < 400 mL/h
Oxytetracycline	Tendon relaxation Antibiotic treatment for *Lawsonia intracellularis*	2.5–3 g per foal diluted in fluids IV q12 h 3–5 days for contracted tendons. 6.6 mg/kg IV (diluted in fluids) q12–24 h for 7 days for *L. intracellularis*
Oxytocin	Esophageal relaxant for treatment of choke Abortigenic for retained placenta Post-partum metritis	0.11–0.22 IU/kg IV following xylazine and flunixin for treatment of choke. 10–40 IU IM q8–12 h (start at lowest dose). 100 IU/L as CRI for unresponsive cases of retained placenta
Penicillin procaine G	Antibacterial for Gram-positive infections	20,000–50,000 IU/kg IM q12–24 h
Pentoxifylline	Vasodilator/rheologic agent Anti-TNF therapy	8.5 mg/kg PO q8–12 h. 7.5 mg/kg IV in 500 mL saline q8–12 h
Pentobarbitone	Anticonvulsant; sedative	2–10 mg/kg q3–6 h (as required) IV

Drug	Indication	Amount, route, frequency
Pethidine	Sedative, analgesic	1.0–3.5 mg/kg IM not IV as sedative 5 mg/kg IV as analgesic
Phenazopyridine HCl	Relief of irritation/spasm of urinary tract mucosa; urinary tract anesthesia for bladder irritation or urethritis. Urine will stain	4 mg/kg PO Q8–12 h
Phenobarbital	Anticonvulsant; sedative; anesthetic	5–10 mg/kg in 30 mL saline as a loading dose slowly IV then 2.5–6 mg/kg maintenance dose q8 h IV or PO
Phenylbutazone	Anti-inflammatory	0.25–5 mg/kg q12 h IV or PO
Phenylephrine HCl	Causes splenic contraction (for nephrosplenic entrapment)	Single dose of 20 mg in 1L saline over 10–15 min
Phenylpropanolamine	Increases urethral tone	1.0–2.0 mg/kg PO q8–12 h
Phenytoin extended release	Anticonvulsant	2.8–16.5 mg/kg PO q8 h
Polymixin B	Anti-endotoxic	3000 IU/kg IV q8–12 h
Prednisolone sodium succinate (Solu-Delta-Cortef®)	CNS/spinal trauma; shock dose	1.0–2.5 mg/kg IV once
Primidone	Anti-convulsant	20–40 mg/kg q12 h PO
Procainamide	Anti-arrhythmic used for VPC, ventricular tachycardia, supraventricular tachycardia	1 mg/kg/min IV up to a total dose of 20mg/kg. Put 20 mg/kg dose in 250 mL saline and administer over 20–30 minutes
Propranolol	β-adrenergic blocking agent; anti-arrhythmic effects, APC, VPC, ventricular or atrial tachyarrhythmias	0.03 mg/kg IV or 0.38–0.78 mg/kg PO q8 h
Propofol	Anesthetic induction agent	2–5 mg/kg IV following pre-med
Psyllium	Treatment of sand impactions	0.5–1 g/100 kg PO q6–24 h
Pyrimethamine (Daraprim®)	Anti-protozoal myeloencephalitis; can be toxic to the fetus in the last trimester	1 mg/kg PO q24 h with potentiated sulfa therapy
Ranitidine	Anti-ulcer, gastric H_2 receptor antagonist	1.4 mg/kg IM or IV q8 h; 6.6 mg/kg PO q8–12 h
Rifampin	Used in combination with other antibiotics to treat *R. equi* infections	5 mg/kg PO q12 h; 10 mg/kg PO q24 h
Romifidine	Sedative	20–70 mg/kg once IV or IM
S-adenosylmethionine (SAMe) Denosyl™	Antioxidant useful for liver disease	20 mg/kg q24 h PO
Sodium polystyrene sulphonate	Used for reduction of elevated potassium levels such as in uroperitoneum in foals	15–30 g/100 kg, PO or enema q12 h

Drug	Indication	Amount, route, frequency
Streptomycin	Antibiotic (used for Leptospirosis)	5–15 mg/kg q8 h IV or IM
Sucralfate	Coats oral, gastric and intestinal ulcers	20 mg/kg q6–8 h PO
Sulfadiazine/trimethoprim	Antibiotic	30 mg/kg q8–12 h IV; 30 mg/kg q12 h PO
Sulfamethoxazole/ trimethoprim	Antibiotic	30 mg/kg q12 h PO
Terbutaline	Bronchodilator	0.06 mg/kg q8–12 h; 10–15 mg (total dose for foal)
Thiamine	CNS disorders	1 mg/kg q24 h IV IM or PO
Ticarcillin	Antibiotic	50–100 mg/kg q8 h IV
Ticarcillin/clavulanate	Antibiotic with clavulanate to block penicillinase	50–60 mg/kg q8 h IV or IM
"Triple drip"	IV anesthesia	1 L 5% guaifenesin + 500 mg xylazine 1 gram ketamine. Maintained at 2–3 mL/kg/h
Valcyclovir	Herpes virus infections	30 mg/kg PO q12 h
Vancomycin	Antibiotic commonly used for *Staphylococcus* infections and resistant *Clostridium difficile*	6 mg/kg q8 h IV slowly 4 mg/kg loading dose then 2 mg/kg PO q6 h
Vitamin C (ascorbic acid)	Antioxidant, enhances vascular integrity	100 mg/kg/day IV
Vitamin E	Antioxidant	8000–15,000 IU PO q24 h; 0.5–2.5 mg/kg IM or SQ q4–6 h
Voriconazole	Antifungal; new triazole useful for *Aspergillus*	3 mg/kg PO q12 h
Xylazine	Sedative	0.2–1 mg/kg once IV or IM
Yohimbine	Treatment for xylazine reversal	0.075 mg/kg IV

Guidelines for drug use in the neonate

Introduction

There are several differences between neonates and adults with regard to the absorption, metabolism and excretion of drugs. Dosage regimens for some drugs are therefore different in the neonate.

The equine neonate is in relation to drug pharmacokinetics a rapid maturer with many metabolic and excretory pathways being close to adult levels by 1 week of age. By 1 month of age foals are similar to adults in most aspects of drug handling.

In the preceding formulary we have given dosages for neonates and adults separately where appropriate.

Selection of antimicrobials

- Almost all systemic neonatal bacterial infections involve gram-negative bacteria, with or without accompanying gram-positive organisms. (The opposite is usually true in adults)
- Bactericidal drugs are also preferred in neonates as many have partial or complete failure of passive transfer in addition to immature/niave defense mechanisms. Therefore an initial antimicrobial selection should consist of bactericidal drugs that cover both gram-negative and gram-positive organisms. Antimicrobial selection should wherever possible be based on sensitivity testing however in many neonates antimicrobial therapy will have to be initiated prior to such results being available. Good initial choices are cephalosporins (cefquinome and ceftiofur) which can be used alone or combined with

aminoglycosides (amikacin and gentamicin). Aminoglycosides can also be combined with penicillin for initial broad spectrum coverage.

- Cephalosporins, aminoglycosides, and penicillins are also excreted unchanged in the urine and undergo limited hepatic metabolism. This is an important consideration as many systemically ill neonates and premature foals will have impaired liver function. Examples of drugs which require extensive hepatic metabolism are macrolides and chloramphenicol.
- There is some concern regarding the use of aminoglycosides because of their potential nephrotoxicity. The following should be considered if using aminoglycosides:
  - Once daily dosing of aminoglycosides is effective and less nephrotoxic than multiple dosing regimens.
  - There are few reported problems in normally hydrated foals. Dehydration enhances toxicity, this is especially important in foals with diarrhea.
  - They should be used with caution (or avoided) in foals with renal disease and premature foals. If used in such foals or if used for prolonged periods, the dosage interval should be adjusted (lengthened) according to measured peak and trough plasma concentrations.
  - Signs of nephrotoxicity should be monitored. Serum concentrations of creatinine can be measured but alterations in the fractional excretion of sodium and increases in the urinary GGT-to-serum creatinine concentration ratio are earlier indicators of tubular damage than is serum creatinine alone. A value of 0.3% + 24 has been published as a reference value for fractional excretion of sodium in healthy suckling foals. The GGT:serum creatinine ratio in healthy perinatal foals should be ≤46.5.

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